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ORIGINAL ARTICLES

SPECIFIC MEDICATION IN TUBERCULOSIS WITH SPECIAL REFERENCE TO CHEMOTHERAPY*

BY EDWARD R. BALDWIN, M.D.

To the physicians here present who are able to look backward over a generation, the following quotation, frequently repeated by the late Doctor Trudeau, will be fully relished:

"The tubercle bacillus bears with equanimity many forms of medication that are fatal to its host!" How truly spoken this thought, and how often forgotten!

It is to bring before you some of the history of tuberculosis medication based on theories of a specific action and their merits or fallacies that I proposed this subject. New cures appear to be the perennial prey of the predatory press which exploits the products of quackery. Nevertheless it is the goal of much honest endeavor, quietly and patiently pursued. It is not to be thought that my purpose is to register the discouragement of our hopes, which thus far remain unfulfilled, but rather to assess or evaluate the accomplishments to date, and to suggest their limitations.

There is a clearer knowledge and a surer basis for treatment than ever before, however much there is yet to be learned. When one recalls the absurdities, not to mention the cruelties, practised on patients in the name of specifics, there is real cause for shame which should give pause to further recklessness with new "cures." The era of hit and miss treatment is passing and the stage of more rational endeavor has been entered. Too frequently we have been urged to experiment on patients before reasonable efforts have been made to find out the value of a given theory by animal experiments which require time.

The past two years have been occupied in a serious effort to discover the action, both on animals and on man, of certain gold preparations. It is a valuable example of what can be done, and I shall refer to the results more in detail later.

For a brief survey of specific medication in tuberculosis in the past we may discern five periods worthy of description:

(1) The pre-bacillary era, before the discovery of the tubercle bacillus. We may call it the period of pure empiricism and it covers all the time before 1882 and for that matter a decade later.

(2) The period of anti-bacillary or germicidal efforts, following the discovery of the bacillus.

(3) The tuberculin period, one that is still continuing to a degree since 1890.

(4) The period of sero-therapy dating from about 1894 and continuing with waning confidence to the present time.

(5) The chemotherapy period, which has been an especially active field for experiment since Ehrlich's discovery of salvarsan.

The forgotten chapters of medical history would contain many former kinds of treatment only vaguely remembered today. Their rationale was also vague. It could hardly be expected that any agreement could exist on the treatment of a disease whose cause and pathology were both unexplained. Most treatments were aimed at symptoms. Consequently inhalations and cough sedatives were in vogue. Hypodermic medication was fortunately little used.

Nevertheless, the heavy metals now used in chemotherapy, like arsenic, mercury and silver, were frequently employed in the past, together with the salts of lime, potassium and phosphorus. The balsamic vapors had repute partly because of the alleged healing effect of pine forests, an ancient belief which fortunately still persists. In the wisdom of our fathers, the judicious use of cod liver oil was rightly valued as a specific. We ought not to dispute the point whether it acts directly or indirectly on tuberculosis.

When the ideas of purging and blood-letting were finally abandoned, I do not know; but these measures hardly deserve the name or were believed to be specific. Alcoholics did, however, achieve a high popularity as specifics, especially in the middle of the nineteenth century, nor have they lost it entirely.

The late Prof. Alonzo Clark was wont to ex-

*Lecture before the Rochester, N. Y., Medical Society, February 2, 1927.

hibit a man to his students, who in spite of, or as he believed, because of, his chronic alcoholism, had recovered from phthisis. In this instance it may be mentioned parenthetically that the man in addressing the students requested them to suggest a cure for alcoholism!

Time fails for the mention of the Indian herbs, the bark tinctures, and many products of polypharmacy, whose virtues were clearly connected with their alcoholic menstruum, like the Deacon's bitters.

When Koch announced the discovery of the tubercle bacillus little time was lost in beginning the search for a germicide that would kill the bacillus without injuring the patient. The idea was attractive and simple. It was also logical, but success was soon found to be far from feasible. Koch himself was the first to see this. While mercury and gold salts were strongly germicidal in cultures or when in direct contact with the bacillus, they were too poisonous and the bacilli too inaccessible in the tissues. Many forms of creosote appeared to be better tolerated than metallic salts and long had popularity as specifics. Experimental support, however, was lacking when they were put to the test in animals. The experiments were crude in comparison with present day standards, yet clinical tests were quite extensive and gave a degree of encouragement for a direct bacillicidal action of creosote derivatives. The essential oils, peppermint, clove, eucalyptus and the like, obtained favor as inhalants or for internal administration on the germicidal theory. Cultures of tubercle bacilli can be arrested in their growth by their vapor, but conditions in the body preclude concentrated action from volatile oils except on the surface of the bronchi. The inhalation of strong vaporized oils has an appeal to the sufferer from pulmonary tuberculosis, as the means of reaching his malady more directly than by other routes. For this reason much charlatanism has been associated with inhaled medicaments, as with electrical methods.

But the merits of all previously known specific treatments were thrown into the background by Koch's tuberculin. Those who can recall the joy with which the discovery was announced in 1890 will also remember the apparently convincing evidence of its specificity. Here was a substance that could be seen under the very eyes to act on the diseased tissue. The skin and larynx could be observed to become violently inflamed under its action, and this was often followed by recession of the foci and their cicatrization.

Here for the first time was observed a truly specific effect produced by a specific agent. The disappointment at the failure of tuberculin does not detract from the major importance of its selective action on a tuberculous focus.

There were other substances found to produce mild focal reactions, notably bacterial proteins, nuclein, and cinnamic acid. These were used therapeutically with the object of promoting the growth of fibrous tissue in and about the tubercles, whereas tuberculin was frankly claimed by Koch to cause the death of the bacilli and the absorption of the tuberculous foci. Just why this claim was unfulfilled may be better understood in my later remarks. Enough truth remained in Koch's original statements of his hope for tuberculin to make it one of the most important observations hitherto noted about tuberculosis.

Here was a disease with focal manifestation to be dealt with, and the cause thereof only to be reached by penetrating or removing the foci. The expectation of accomplishing this end when caseation had already taken place might be unreasonable; nevertheless, cicatrization and encapsulation would be promoted. Beyond question such results did follow tuberculin treatment and can be obtained in localized disease such as in the eye, skin, larynx and occasionally in the lungs. The risk of mobilizing the bacilli and spreading the disease has less importance than the danger of causing complications like hemoptysis, high fever and interference with nutrition, which may in turn secondarily aggravate pulmonary tuberculosis. Therefore, however useful in principle tuberculin may be, its employment is restricted to foci that can be directly watched under the eye or to those pulmonary cases which fail to heal though well localized. Until some new method of controlling reactions is known, the dosage must remain empirical and experimental.

In all that I have said concerning tuberculin it is to be inferred that without focal reactions of some degree this treatment is virtually without influence on the disease, albeit some good may result by lowering the state of sensitiveness to reactions; this at least is my own opinion from theoretical grounds. Nothing superior to tuberculin has yet been contributed to act specifically on tuberculous tissue—and by tuberculin I mean any preparation from tubercle bacilli containing the reaction-producing substance.

Unceasing efforts to improve tuberculin by modifications, or combination with other substances aimed at the bacilli, are still being made. It is theoretically desirable to introduce a germicide simultaneously with a tuberculin reaction, and when the blood and lymph flow are most intense in the foci.

Success in this object is yet unattained, but the indications for rational therapy in tuberculosis became clearer during the tuberculin era.

Following close upon tuberculin came the antitoxic sera for various infectious diseases. It would be profitless to relate the story of efforts toward such a treatment for tuberculosis. Suffice it that no really scientific proof of anti-

toxic substances in the blood or tissues of tuberculous animals has been produced. Positive results in experiments are capable of other explanations. Any foreign protein, even homologous blood, has a certain nonspecific effect, besides its nutritive action, on another animal. Heterologous blood or serum excites as we know the formation of substances capable of precipitating the foreign serum protein, and producing reactions known as anaphylaxis in highly sensitized subjects, after repeated injections of serum. Thus serotherapy of any kind may cause a nonspecific stimulus, indirectly assisting the body to combat disease. This is, however, far from the neutralizing toxin-antitoxin conception. It must be acknowledged that a few instances of blood transfusion from convalescent to actively tuberculous individuals have appeared to be directly protective against infection, but they are isolated cases.

All sorts of gland products and tissue extracts have been exploited for tuberculosis without adequate or controlled evidence of their specific influence. It is natural to think that since whatever defense we possess against tubercle bacilli appears to reside in the cells of the body, we should expect it to be susceptible of transfer to others in the form of an extract. The secret is, nevertheless, as yet unrevealed, and awaits further knowledge of Nature's physiological laws of adaptation to the offending bacilli. Certainly we have no biological product capable of neutralizing the symptoms of fever, sweats and nervous exhaustion accompanying progressive tuberculosis, if such be our aim.

The more recent claims of Mr. Spahlinger of Geneva, Switzerland, who has made incessant efforts for recognition in Europe, may at the best be classed as honest delusions. They also illustrate the fallacy of depending on clinical improvement in chronic tuberculous patients. Spahlinger's own statements about the form of treatment used easily place it in the class of tuberculin modifications combined with the use of various sera alleged to be good for the symptoms of acute disease. Enough is known of the method of preparing these substances to indicate the futility of subjecting them to a scientific test.

A thorough chemical and physiological investigation of the tubercle bacillus and its ingredients is now in progress with the financial aid of the National Tuberculosis Association of our country. This will do much to pave the way for biological research into the deeper problems of specific immunity or to confirm what we already know from analogy.

The fluctuations of bodily resistance due to a thousand influences must be reckoned with when we talk of conferring immunity to tuberculosis. It is therefore without doubt that the ideal specific against tuberculosis would be a substance or method that could slay the microbe on sight, so to speak, and like quinine in malaria, be able to scotch the bacilli whenever they emerge from their hiding places if already concealed in the body as they generally are. This conception has never ceased to be the object of experiments, but during the past fifteen years efforts have been multiplied because of Ehrlich's brilliant success with spirochetal diseases.

Chemotherapeutic research has been a prominent part of the recent work done in pharmaceutical laboratories, and the literature teems with new synthetic remedies for all infectious diseases. While there are new aspects of chemotherapy, in the main the object seems to be to make some compound with an affinity for the given microbe and containing a poisonous fraction, usually arsenic. The aniline dyes first came into use for these compounds. The success which has been obtained with dyes coupled with arsenic, or others without such combination, with malaria and to some degree with pathogenic cocci, could not be matched in tuberculosis. Methylene blue, Nile blue, gentian violet, trypan red and others like trypanflavin and rivanol, which are useful in streptococcus infections, fail to influence tuberculosis in animals. Many have been tried, but since we know how difficult it is to penetrate the tubercle bacillus with dyes, the failure is not surprising. Owing to the waxy character of the protoplasm the acidfast bacilli require a mordant to carry the stain into their substance. Even with this aid, the stain acts slowly unless heat is used. Cresols, among organic agents, are possibly the most active germicidal substances against the tubercle bacilli outside the body, but they lose their potency when in contact with animal tissues, in addition to being quite poisonous.

Attempts were made at one time to treat pulmonary tuberculosis by intravenous injections of formalin to the point of toleration. The bacilli were not killed, but the patients barely survived.

With the passing of the years a succession of inorganic compounds have appeared and disappeared. Iodine possibly has enjoyed a long and honorable repute for tuberculosis, especially for the lymphatic type. The other halogens, bromine, chlorine and fluorine, have found advocates, but in the combinations required to make them safe, no direct germicidal effect is likely to occur. In the last decade an increasing interest has developed in the compounds of copper, mercury, silver and particularly gold. It almost appears to be a return to the medicine of the ancient Arabian alchemists, who searched for the elixir of life by the transmutation of base metals into "potable gold."

Shakespeare, whose familiarity with medical traditions was little short of marvelous, refers to "potable gold" in "King Henry IV," where Prince Henry addresses the gold crown which

he is soon to inherit from his careworn, invalid father:

"The care on thee depending
Hath fed upon the body of my father;
Therefore, thou, best of gold, art worst of gold;
Other, less fine in carat, is more precious,
Preserving life in medicine potable." *

Since at the present time we are witnessing a revival of interest in therapy by metallic salts, it may be of interest to go into some details of their action as so-called chemo-specifics for tuberculosis. Koch himself found that gold cyanide could restrain the growth of tubercle bacilli in culture in 1 part to 1,000,000, but in blood serum only in 1 part to 25,000.

Gold chloride is equally powerful, but both were found ineffective in animal disease. Copper salts also inhibited the growth of bacilli in small quantities. Some years ago the German Countess von Linden made enthusiastic claims for copper treatment, especially in lupus. More recently Koga of Japan introduced copper cyanide under the name of cyanocuprol. The results of intravenous treatment with cyanocuprol failed to confirm the hopes of the author, and furthermore it was dangerous.

More recently a variety of gold preparations have appeared and been given a clinical trial. Such are gold cantharidin, called aurocanthan, krysolgan and triphal. All these inhibit the growth of the tubercle bacillus in cultures to varying degrees, but produce objectionable reactions, both focal and general, when used in the living body.

Still more recently Mollgaard, a Danish veterinary physiologist, made far-reaching claims for the selective affinity of gold sodium thiosulphate for tubercle bacilli. During the past two years many tests have been made of "Sanocrysin" on laboratory animals and on patients, with conflicting results.

It may be said that for the first time since the introduction of tuberculin a scientific inquiry has been encouraged by the author of a remedy for tuberculosis. The work has been carried on in this country chiefly by the United States Hygienic Laboratory and at the Chicago Municipal Sanatorium, but the preparation has not been used to any extent on patients.

Inasmuch as reputable experimental evidence has been adduced both for and against the use of sanocrysin, the basis of this remedy seems worthy of discussion. I venture to do this since it brings out quite clearly certain misconceptions very prevalent as to what tuberculous disease really comprises in its entirety. Furthermore, as we are prone to look upon serious laboratory experiments with respect, it is our duty to know whether application of the so-called specific in question can be made to human disease.

In the first place, sanocrysin has been said to inhibit the growth of tubercle bacilli in culture in the same minute proportion that characterized potassium gold cyanide: viz., 1 to 1,000,000. When this statement was put to the test, it was found that much stronger concentrations were needed. Even 1 to 1,000 was tolerated in liquid cultures, and a 1 per cent solution failed to kill all the bacilli after 24 hours' immersion. Moreover, no evidence has been produced that the gold salt enters into combination with the bacillus.

Secondarily, the salt when administered intravenously produced well marked focal reactions in the tuberculous tissue, followed by fever due, it was thought, to the death and dissolution of tubercle bacilli in the lesions, thus setting free endotoxins. The implication was that a direct action on the bacilli occurred, which in the light of the before-mentioned facts appears unlikely. Moreover, in animal experiments it was shown that the bacilli were not all killed.

In the further development of the treatment a so-called antitoxic calf or horse serum was employed to neutralize the shock of the focal reaction. I shall only refer to this as another instance of non-specific supporting treatment without proof of antitoxic action.

Thirdly, it was found that only early, moderately extensive cases of consolidation of the lung were suitable for treatment and that only a few who were treated lost the bacilli from the sputum.

The clinicians in general agreed that careful selection of cases must be made because harm could be done to chronic pulmonary types and especially those complicated by intestinal disease.

Finally, it had been stated that metallic poisoning affecting the kidneys, skin and mucous membranes, which is characteristic of gold and mercury, could be avoided by proper dosage. This has not been true in all cases.

A critical summary of the literature on sanocrysin does not therefore give any ground for the high hopes held out by Mollgaard. From the standpoint of a specific, there is but one element that arrests attention, and that is the focal reaction in the tuberculous tissues. For this, there is no longer reason to think a specific agent is necessary. It has long been known that many substances administered intravenously excite vascular congestion in recent tuberculous tissue. Evidence of the toxic action of gold on the capillary endothelium (Heubner) has been known. This is sufficient to account for the exudation and inflammation, and chronic inflammatory foci are particularly susceptible to irritation. There is also much reason to regard the action of gold as a catalyzer increasing the rate of oxidation or ferment action in the tissues. If tubercle bacilli are destroyed in the process it is not necessary to assume that gold *per se* is the agent. The important observation is that tuber-

*King Henry IV, Second Part: Act IV, Scene iv.

culous tissue becomes absorbed more quickly and secondarily tubercle bacilli are digested or otherwise disposed of by the physiological processes of the body. All the studies that have been made of the distribution of gold in the body have not found a concentration in the tuberculous tissues or lungs, but more in the kidneys than elsewhere.

Thus another fallacy has been overthrown and together with it the idea that the severe reactions are solely due to tubercle bacilli being disintegrated. Metallic poisoning has produced quite similar effects on normal animals, so must aggravate whatever tuberculin poisoning may occur. The elimination of gold is slow. Only half that injected is excreted promptly, chiefly by the kidneys, and the remainder is got rid of very gradually so that a cumulative action is only to be avoided by constant test of the urine.

The preponderance of adverse opinion, outside of Denmark, will soon restrict the use of sanocrysin, but we may draw some valuable lessons from this latest respectable contribution to tuberculosis treatment. Several questions naturally arise:

(1) Is it desirable to attempt to kill tubercle bacilli in the body, in the face of the fact that they are already embedded in the tissues and only occasionally get free in the lymph or blood circulation? I believe it possible to conceive of a harmless substance placed in the circulation frequently enough to meet all escaping bacilli and render them powerless, if not to destroy them; but to attempt to break down tuberculous tissue already of considerable extent is fraught with danger, and when fibrous, is irrational.

(2) Is any agent acting upon tubercle like tuberculin likely to be superior? I think not, for experience shows that for internal tuberculosis at least, any agent causing inflammatory reactions has no obvious advantages. It is possible that some germicide combined with tuberculin may work to our advantage, yet experiments along this line have not accomplished much thus far.

The most hopeful methods of treatment yet devised involve indirect action, except in the external forms of tuberculosis which are accessible to local treatment. The absorption of recent inflammatory or so-called collateral inflammation surrounding a tuberculous focus is often complete without any treatment other than rest and good hygiene. If the added stimulus of focal reactions should be needed to hasten the absorption the means used must be safe or applied with the discretion born of experience. In truth, the first thought concerning any specific for tuberculosis should be, can it improve on Nature?

For the present, the theory that a specific has been found to act directly on the bacilli has lost favor. Instead, the best explanation of the action of tuberculin and similar irritants is that they hasten and intensify oxidation and enzyme activity, which in turn affect the bacillus. Possibly the best illustration of this is furnished by the often remarkable effect of natural sunlight or artificial radiation on lupus and surgical tuberculosis as well as upon intestinal complications in pulmonary disease. There are also recently published experiments (Walbum) pointing to a marked stimulation resulting from the injection of minute quantities of mineral salts, especially manganese. The author regards them as catalyzers or substances that increase enzyme and hence immunizing functions. Exact concentrations of the salts are required according to molecular weight in order to achieve the best results. There is an optimum concentration for each salt which follows the law of Arndt, which is:

Weak irritants arouse vital action, medium strengths promote it, but strong doses decrease, and the strongest doses abolish it.

Thus Nature provides for the equilibrium of our cells, that it shall not be unduly upset by changes in the mineral constituents and hydrogen ion concentrations of the blood fluids.

The subject of chemotherapy is undoubtedly fascinating and the field is open for new ideas. Let us meanwhile be pleased to assist the natural weapons provided for our defense.

THE EFFECT OF OPERATION UPON THE VITAL CAPACITY*

BY JEROME R. HEAD, M.D.†

In consideration of the etiology of post-operative pulmonary complications, increasing importance is being attached to the effect of operations upon the mechanics of respiration. Since 1876, when Pearson-Irvine[1] reported the first instance of collapse of the lungs secondary to paralysis of a group of the muscles of respiration, it has been known that a localized restriction of expansion of the chest wall could produce pathological changes in the subjacent lung; and since 1908, when William Pasteur[2] noted the first instances of what he termed post-operative "massive collapse of the lungs," it has been recognized that operations, especially upon the abdomen, frequently occasioned sufficient thoracic and diaphragmatic immobility to produce the same results.

The cases reported by these writers were of true massive collapse or atalectasis. Pasteur recognized that this was an extreme condition and that there are many lesser degrees of the same process. These are not easily recognized clinically, but being infinitely more frequent and predisposing equally to pneumonia, they are more important. Pasteur[3], among his cases of post-

*The data upon which this paper is based were collected during the year 1925 while the author was resident in Surgery in the State of Wisconsin General Hospital. The paper was presented before the Research Club of the Medical School of the University of Illinois on December 22, 1926, and was submitted for publication on February 8, 1927.

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diphtheritic diaphragmatic paralysis, noted instances of small scattered areas of collapse. Briscoe[4] found them in rabbits after section of the phrenic nerves. Lee[5] cites Paul to the effect that such areas are very frequent in the lungs after death. These findings have suggested that post-operative restricted respiration is more important than has formerly been believed and probably accounts for a much greater number of pulmonary complications than the eight per cent estimated by Pasteur.

In view of these facts, it seemed probable that a study of the effect of operation upon the vital capacity would give interesting data, would provide a rough quantitative estimate of the degree of interference with respiration and perhaps explain why, following certain operations, pulmonary complications have a high incidence. Consequently such studies were made upon a group of patients who had undergone operations for a variety of conditions. Readings were taken before operation until a constant normal was established. Following operation they were taken each day until the capacity had returned to the pre-operative level, or until a new level had been reached, or the patient was discharged. Great pains were taken to interest the patients in the examinations, and to stimulate the spirit of competition. They were encouraged to exert themselves in spite of pain. Each day the highest of three trials was recorded. The results are shown in the following charts:

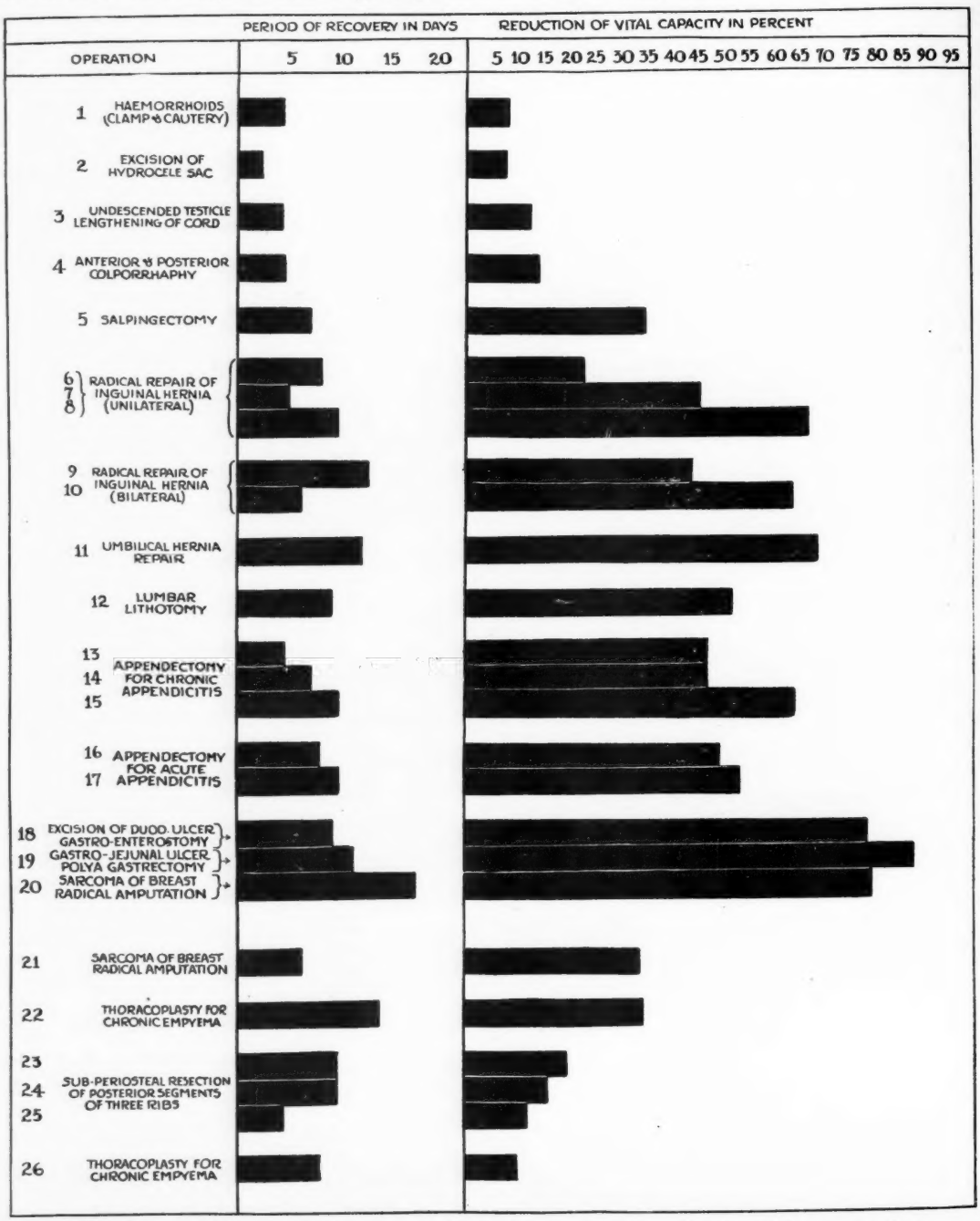
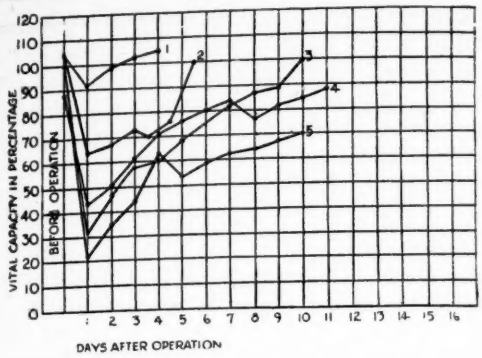
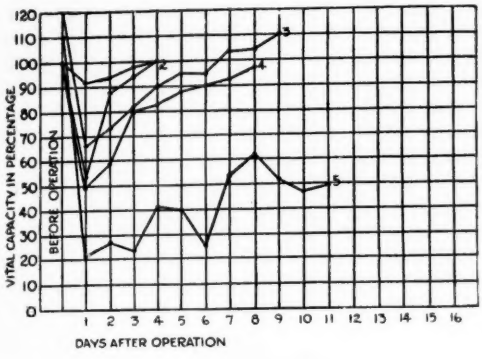

PERIOD OF RECOVERY IN DAYS
REDUCTION OF VITAL CAPACITY IN PERCENT
OPERATION
5 10 15 20
5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95
1 HAEMORRHOIDS (CLAMP & CAUTERY)
2 EXCISION OF HYDROCELE SAC
3 UNDESCENDED TESTICLE LENGTHENING OF CORD
4 ANTERIOR & POSTERIOR COLPORRHAPHY
5 SALPINGECTOMY
6 7 8 RADICAL REPAIR OF INGUINAL HERNIA (UNILATERAL)
9 10 RADICAL REPAIR OF INGUINAL HERNIA (BILATERAL)
11 UMBILICAL HERNIA REPAIR
12 LUMBAR LITHOTOMY
13 14 15 APPENDECTOMY FOR CHRONIC APPENDICITIS
16 17 APPENDECTOMY FOR ACUTE APPENDICITIS
18 EXCISION OF DUOD. ULCER GASTRO-ENTEROSTOMY
19 GASTRO-JEJUNAL ULCER POLYA GASTRECTOMY
20 SARCOMA OF BREAST RADICAL AMPUTATION
21 SARCOMA OF BREAST RADICAL AMPUTATION
22 THORACOPLASTY FOR CHRONIC EMPYEMA
23 24 25 SUB-PERIOSTEAL RESECTION OF POSTERIOR SEGMENTS OF THREE RIBS
26 THORACOPLASTY FOR CHRONIC EMPYEMA

PLATE 1. Graphic representation of the reduction in Vital Capacity produced by different operations.



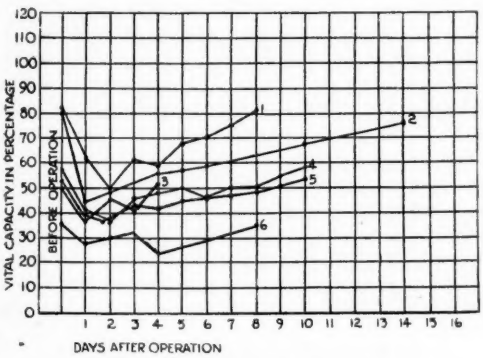
1. Undescended testicle - Lengthening of cord.
2. Salpingeotomy
3. Inguinal hernia - Unilateral
4. Inguinal hernia - Bilateral
5. Duodenal ulcer - Excision and gastro-enterostomy

PLATE 2. Chart showing the changes in Vital Capacity from day to day following a typical group of operations.



1. Haemorrhoids - Clamp and Cautery
2. Ureteral Stone - Lumbar Lithotomy
3. Chronic Appendicitis - Appendectomy
4. Acute Appendicitis - Appendectomy
5. Cancer of Stomach - Exploration

PLATE 3. Chart showing the changes in Vital Capacity from day to day following a typical group of operations.



1. Carcinoma of Breast - Radical Amputation
2. Chronic Empyema - Thoracoplasty
3. Subperiosteal Resection - of segments of three ribs
4. Subperiosteal Resection - of segments of three ribs
5. Subperiosteal Resection - of segments of three ribs
6. Chronic Empyema - Thoracoplasty

PLATE 4. Chart showing the changes in Vital Capacity from day to day following a group of operations upon the thorax.

An analysis of the curves brings out the following points:

1. In all instances there was a post-operative fall in vital capacity, followed by a gradual return to normal. The reduction varied in the different cases between five and eighty per cent and the period of recovery between one and eighteen plus days.

2. After operations not involving incision of the muscles of respiration the reduction was slight and the return to normal rapid. Thus after clamping and cauterization of haemorrhoids, the reading fell from 100 to 92 per cent (eight points) and then gradually rose to regain the normal level on the fourth day after operation. After an anterior and posterior colporrhaphy the decrease was twelve per cent and the period of recovery four days. After lengthening the cord in undescended testis (without section of the external oblique) the figures were the same; after excision of a hydrocele sac the reduction was twenty per cent and the pre-operative level was regained in two days.

3. Following operations involving incision of the muscles of respiration the reductions were greater and the periods of recovery more protracted.

4. Abdominal operations produced the greatest changes. In these the reduction in vital capacity was usually proportionate to the extent of the procedure and the proximity of the field to the diaphragm.

5. The amount of reduction was determined more by the size and location of the abdominal incision than by the intra-abdominal manipulation.

Points worthy of note are:

a. The results were practically the same for unilateral inguinal hernia, bilateral inguinal hernia, chronic appendicitis, acute appendicits and umbilical hernia—all of them operations upon the lower abdomen.

b. The greatest reductions (78 to 88 per cent) followed operations on the stomach in which the incision extended into the epigastric angle.

c. The reduction was as great following a simple epigastric exploration as following gastroenterostomy or gastrectomy through a similar incision.

d. In none of these latter cases had the reading returned to normal at the time the patient was discharged from the hospital—eighteen days in one case.

6. Following operations upon the chest wall (not involving collapse of the lung) the reductions were uniformly less than those following abdominal operations. Thus the average fall following subperiosteal resection of posterior segments of three ribs was fourteen per cent. After an extensive thoracoplasty for chronic empyema it was only thirty-four per cent.

7. In these cases it was evident that the reduction was less where the original capacity was low. Thus, while a thoracoplasty for empyema on a patient with an initial reading of eighty per cent produced a fall of thirty-four per cent, an equally extensive procedure on a patient whose pre-operative capacity was thirty-five per cent of normal occasioned a drop of only ten per

cent. This would seem to indicate that while no great effort is made to preserve the upper ranges of the vital capacity, reduction below a certain minimum is strongly opposed.

8. In nearly all instances the length of the period of recovery was proportionate to the initial drop.

9. In uncomplicated cases the curve of the return to normal was practically a straight line. Complications, such as wound infection, etc., were shown by irregularities in the ascent.

DISCUSSION

It must be recognized that the vital capacity is only a rough measurement of the effect of operation upon the respiration. Being a maximum gross reading, it fails to indicate the state of quiet breathing and the relative aeration of different lung areas. Other data, the known pathological changes which occur, and a consideration of the nature of the interference indicate that these are altered and that the changes noted in the vital capacity reflect proportionate ones in tidal breathing, alterations which are occasioned by marked under function of certain portions of the lungs.

From a theoretical point of view, operation could reduce the vital capacity either by producing an actual reduction in air space or by limiting the movement of the chest wall and the expansion of the subjacent lung. The air space could be reduced either by passive congestion secondary to cardiac insufficiency or pulmonary immobility, by inflammation of the epithelium caused by irritation of the anesthetic, or by actual mechanical collapse or destruction of pulmonary tissue. Movement of the chest wall could be limited either by pain or by reflex inhibition of the respiratory muscles.

There is no reason to believe that reduction in air space is an important factor. In none of the operations was there a collapse or destruction of pulmonary tissue. Neither before, during, or after the operation did any of the patients have signs or symptoms of cardiac insufficiency; and that the anesthetic was not responsible is evidenced by the fact that many of the procedures were performed under local anesthesia, and that in these instances the reduction was as great as after ether, nitrous oxide or ethylene.

Whether the inhibition is voluntary because of pain or reflex is a matter of discussion. The majority of evidence is in favor of the former while, to support the latter, there is little but supposition. All of the abdominal muscles take an active part in respiration and all of the viscera are passively affected by it. It is reasonable to believe that after operation descent of the diaphragm is inhibited because it produces painful movement of the involved viscera and because by raising the intra-abdominal pressure, it puts increased tension upon the wound; also that inspiration is further restricted because of the same effect upon the wound incident to the upward and outward movement of the lower ribs. If the incision has extended into the critical epigastric triangle, this factor is particularly important. This is reflected in the clinical observation of the increased incidence of pulmonary complications following upper abdominal operations and in the above statistics on the vital capacity. The strength of the lateral pull produced by descent of the diaphragm and by the inspiratory spreading and elevation of the ribs can be estimated by whoever has attempted to close a tight abdomen and found that only on extirpation could the wound edges be brought together.

Pasteur[6], Rose-Bradford[7], Harries[8] and others are of the opinion that reflex inhibition plays an important part. It is well known that movement of the diaphragm can be so curtailed by nonpainful pulmonary pathology. There is, however, no definite evidence that the reflex can be initiated from below the diaphragm. Herries, commenting on the elevation of the diaphragm secondary to intra-abdominal inflammatory lesions, suggests that, being antagonistic to the abdominal muscles, it is reflexly relaxed when these are spastic. That this explanation is not correct is evidenced by the fact that in massive collapse of the lungs the abdominal muscles are regularly flaccid.

Pasteur writes, "The exact nature of the genesis of post-operative collapse must, for the present, remain a matter of speculation. As already stated, there is no evidence that post-operative protective limitation of diaphragmatic movement ever leads on to collapse, which, if due to that cause, would of necessity be bilateral. On the other hand we may conclude with certainty, from the evidence brought forward, that failure of respiratory force is the essential cause of massive collapse. We are, therefore, led to the conclusion that post-operative massive collapse is the result of (reflex) arrest of action of one-half of the diaphragm.

"On this hypothesis, a stimulus of unknown nature arising in one-half of the body reflexly arrests the distending action of the corresponding half of the diaphragm, to such a pitch that the "compressors" of the lower lobe of that side take charge, causing it to collapse in the manner described. One may hazard the conjecture that the afferent path of the reflex is along some branch of the vagus."

Briscoe, by various methods of irritation of the lung, pleura, pericardium, peritoneum and abdominal viscera, was unable to produce any changes in the movement of the diaphragmatic segments. From these experiments he concluded that Pasteur's conception of reflex inhibition was erroneous, also that "the theory put forward by Pasteur that deflation of the lower lobes was dependent upon paralysis of one or

both halves of the diaphragm seemed to be untenable."

He believes that "massive collapse of the lower lobes is the natural sequence of prolonged quiet breathing in the supine position in such people as do not use the abdominal muscles to fix the chest." According to his observation the function of the costal and crural portions of the diaphragm are different, the former acting to expand the lower lobes and the latter acting through the pericardial ligament to lower the hilus and so expand the apices. Since the costal portion acts only when the lower ribs are fixed by the abdominal muscles, and since the supine position tends to prevent this, inefficient aeration and collapse of the portions of the lungs dependent upon this movement results.

Whichever of these theories is correct, it is apparent that the brunt of the respiratory embarrassment is borne by the lower part of the chest. It is well known that, because of the resistance offered by the pulmonary tissue, alterations in the movement of any part of the thoracic cage affect most the lung which is immediately subjacent, and that for their efficient aeration, the different parts of the lung are dependent upon the movement of the corresponding portions of the chest wall or diaphragm. Thus when, as after operation, the action of the diaphragm and lower ribs is restricted, the adjoining lung is immobilized and underaerated more seriously than the other parts.

Clinical and experimental observations have shown that such immobilization produces congestion and oedema, stagnation of secretion and may lead to collapse (atalectasis) and pneumonia. It seems probable that the reduction in vital capacity following operation is a reflection of such localized basal under function.

Since this paper was submitted for publication there has appeared in the April number of *Surgery, Gynecology and Obstetrics* an excellent article by Churchill based upon similar data. In the present article there are, I believe, sufficient additional observations and conclusions to make it worth presenting.

BIBLIOGRAPHY

1 Pearson-Irvine: Trans. Clin. Soc. London, 1876, p. 194.
2 Pasteur, William: Massive Collapse of the Lung. Lancet, 1908, II, 135.
3 Pasteur, William: Respiratory Paralysis After Diphtheria. Am. Jour. of Med. Sc., 1890, C, 242.
4 Briscoe, J. C.: The Mechanism of Post-Operative Massive Collapse of the Lung. Quart. Jour. Med., 1920, XIII, 293.
5 Jackson, C., and Lee, W. E.: Acute Massive Collapse of the Lung. Ann. Surg., 1925, LXXXII, 364.
6 Pasteur, William: Massive Collapse of the Lung. Brit. Jour. of Surg., 1914, I, 587.
7 Rose-Bradford: Massive Collapse of the Lung. Quart. Jour. Med., 1918-19, XII, 127.
8 Harries, D. J.: The Influence of Abdominal and Thoracic Lesions on the Respiratory System. Practitioner, London, Apr., 1923, p. 188.

REPORT OF A CASE OF EVENTRATION OF THE LEFT DIAPHRAGM

BY FRANK E. WHEATLEY, M.D.

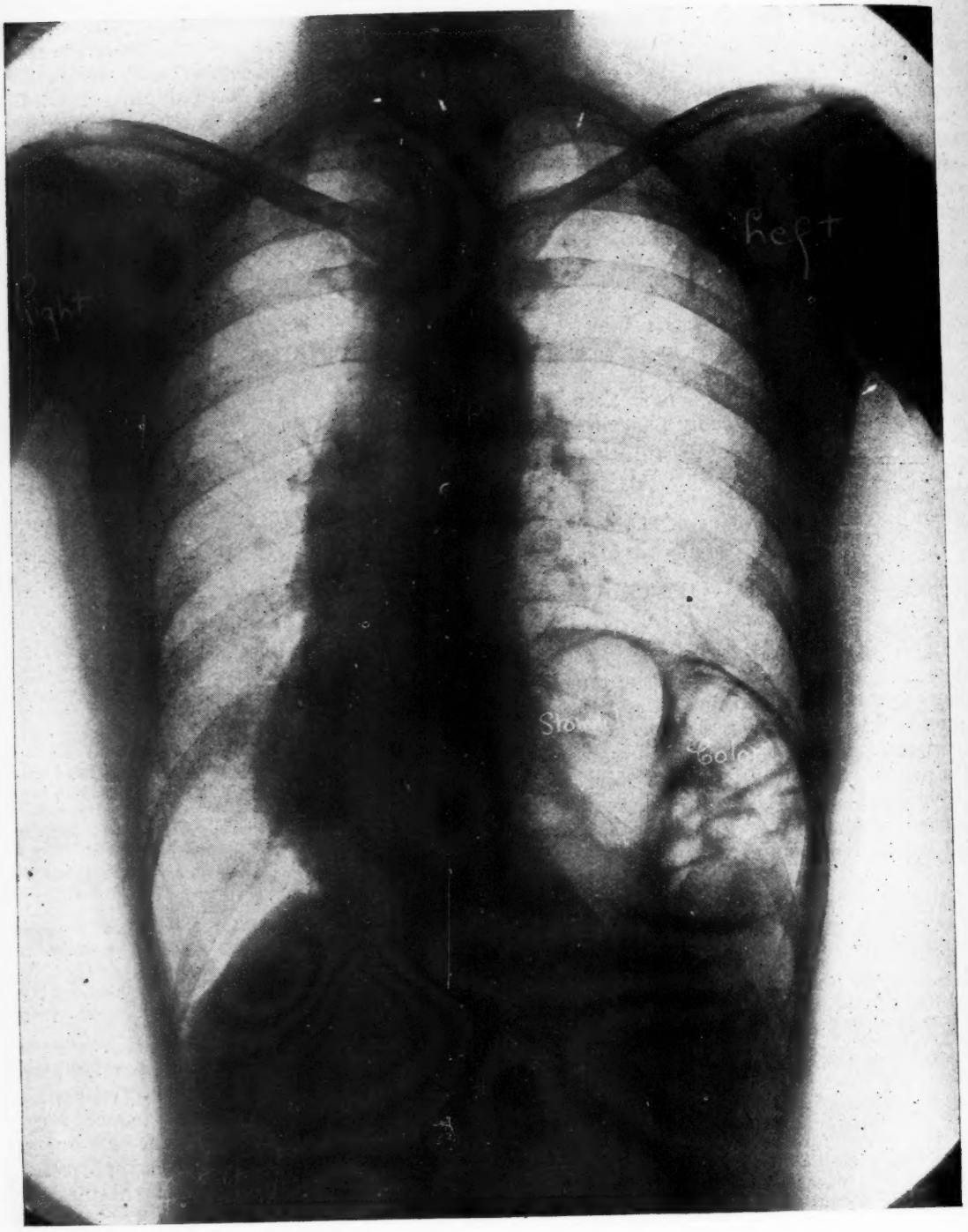
THIS case is reported, not for the purpose of adding anything of scientific value to the investigation of this condition, which has been studied so thoroughly in the last few years, particularly in the papers written by Dr. L. B. Morrison and his associates. It is, however, interesting from a clinical aspect because of the symptoms of which the patient complained and the fact that she had been thoroughly examined at several reputable clinics in Europe, without definite diagnosis and apparently without suspicion of the underlying condition.

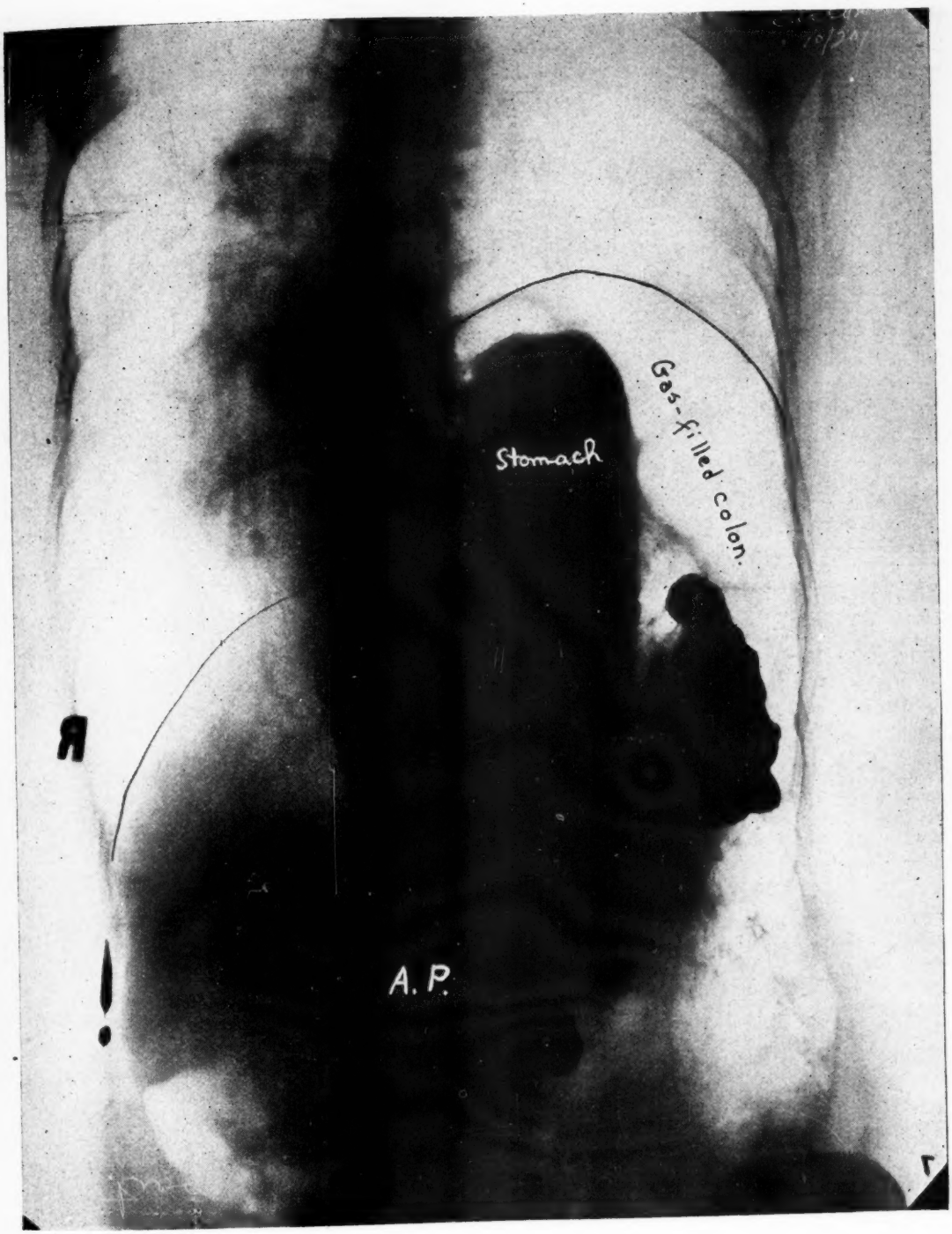
The patient, a woman of 50, developed, within a year, palpitation of the heart on exertion, progressive weakness and indefinite pain extending from the right hypochondrium, through the chest and down the right arm. She also became constipated and suffered from flatulence. Clinical examination revealed no finding of importance, except possibly the heart dullness, extending further to the right than usual. She was seen by Dr. E. N. Libby, who referred her for X-ray examination.

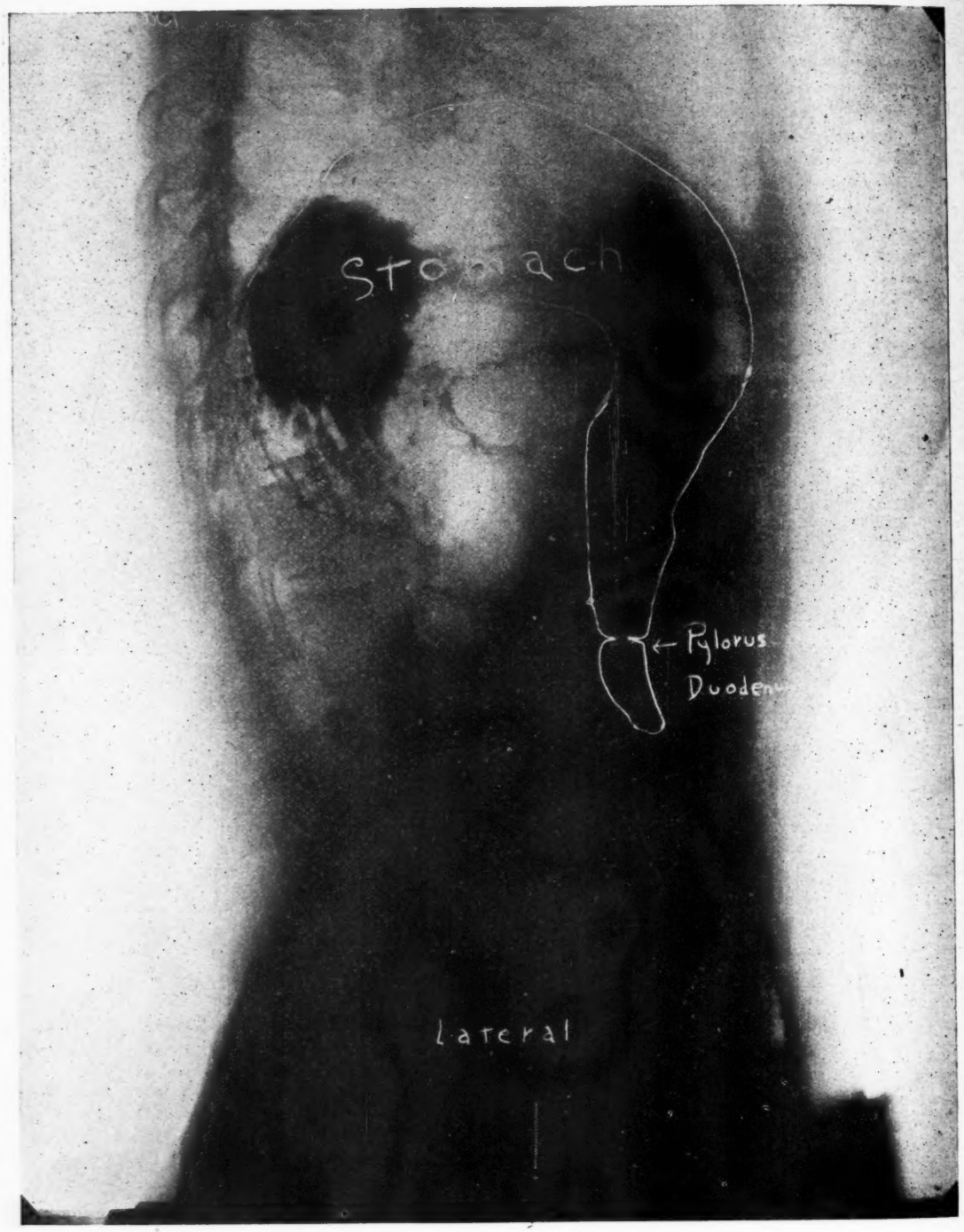
The fluoroscopic examination of the chest showed the heart and mediastinal contents displaced to the right about 4 cm. The right diaphragm was low, with normal excursion. The left diaphragm was at the level of the fifth rib and showed restricted excursion. Beneath the left diaphragm appeared gas-filled viscera, which proved to be the stomach and splenic flexure of the colon. On the administration of barium the oesophagus appeared tortuous and entered the stomach considerably below the highest point of the diaphragm. In the antero-posterior view, the stomach appeared to have two sacs, one posterior to the other. In the lateral view, the posterior sac was shown to be the cardiac portion of the stomach, the pars media being the highest portion of the stomach and supported probably by the gas-filled gut below it. The pyloric portion of the stomach was vertical and the first portion of the duodenum was directly beneath it. The small intestines exhibited no remarkable features. The colon showed depression of the hepatic flexure, probably due to the descent of the liver under the right diaphragm. The transverse colon displayed unusual obliquity upward to the splenic flexure, which was markedly distended with gas and situated to the left of the stomach, reaching the highest point of the left diaphragm. The descending colon, sigmoid and rectum were normal.

A clearer idea of the departure from normal anatomy may be gained by reference to the accompanying plates.

Discussion: This case was particularly interesting because of the difficulty in establishing reasonable cause for the patient's symptoms from physical examination. The reason for the difficulty of determining abnormality in the chest, except for the apparent enlargement of the right side of the heart, is probably the gas-filled viscera beneath the left diaphragm, which not only gave normal resonance to the lower portion of the left chest but which also transmitted sounds by resonance. The displacement of the apex of the heart may have been overlooked at each examination because of the dullness obtained, possibly from food in the stomach or feces in the colon. Although the patient


Right
Left


Stomach
Gas-filled colon
R
A. P.


Stomach
Pylorus
Duodenum
Lateral

gave a history of a serious automobile accident two years prior to the onset of symptoms, it is difficult to believe that the condition discovered was in any way attributable to trauma. It is far more likely that this congenital defect had not caused symptoms because the digestive function in youth and middle age was normal in spite of the handicap, and that advancing age, with a general lowering of tone and digestive secretions, resulted in fermentation and flatulence which would explain practically all the symptoms.

The case is probably parallel to that of an individual who is able to carry on with a faulty heart, but who eventually comes to grief because of failure of compensation. It argues for the advisability of supplementing careful physical examination with X-ray studies in cases where the symptomatology is distressing and the cause is obscure.

TREATMENT OF ANKLE SPRAINS*

BY CHARLES P. HUTCHINS, M.D.

PRELIMINARY to a consideration of the treatment of ankle sprains, the subject which this paper is designed to discuss, the pathological possibilities are first inventoried.

Simple sprain is a rather loose expression applied to injuries to the joints in which the ligaments, tendons or synovia are involved, but no fracture or dislocation of the bone exists; it is the result of extension of motion beyond normal limits. (Ochsner.)

Sprain fracture is the separation of tendon or ligament from its insertion, the bone to which the tendon or ligament is attached being separated from the bone shaft either from direct or indirect violence. (Cotton fracture.)

In incomplete dislocation with spontaneous reduction the capsule generally remains intact. External ligaments are prone to rupture, joint cartilages may be contused and the traversing tendons torn from sheaths, displaced or actually ruptured. Again, articular surfaces may be torn from underlying bone or fracture.

Sprain of the ankle is most often caused by unguarded movement by which the foot is turned suddenly inward, rarely outward, with sufficient force to rupture the ligaments and possibly some of the fibers of the muscle and to strain tendons and sheaths. In the slighter degrees of sprain the injury may be confined to the tissues about the joint, but in some instances there is effusion within the capsule.

We are concerned chiefly with ankle sprains with inversion and eversion—and particularly with the former. Statistics are scarcely necessary, but a long experience with these injuries has impressed all of us with the preponderance of sprains of inversion. This is reasonable when one contemplates the tissues that limit movement. To prevent eversion we have, beside the powerful deltoid ligament, both the tibialis anticus and posticus; these are much stronger than the peroneus longus and tertius, the latter of which is at times lacking. Regard the external ligaments and their directions; the anterior and posterior fasciculi are located better to limit sliding of the joint forward and back than to hold during forcible inversion. This leaves the middle fasciculus to limit the action with the aid of the peronei—and the line of least resistance is plain.

Many years experience with foot-ball injuries has shown me no single instance of anterior or posterior sprain of the tibio-astragaloid joints. It is a joint of high resistance in comparison with tarsal and metatarsal joints. Plantar overflexion is prevented not only by the anterior ligament but by the extensors of the toes and tibialis anticus.

Over-extension is limited, not only by the weak posterior ligament, but by the powerful gastrocnemius, soleus, flexor longus digitorum and flexor hallucis; the tibialis and peroneal muscles, working in opposition to one another, by combined and synchronous action, exert a ligamentous action to limit over-extension and flexion.

When the foot is at the equilibrial angle—a right angle to the lower leg—or when it is dorsally flexed to a lesser angle than that, sprains at the ankle joint are rare. The balance of muscle action seems to support the ligaments sufficiently to hold even in the presence of force almost sufficient to cause fracture of the malleoli. This is a significant point in treatment.

In the absence, then, of unusual features of concomitant fracture, split cartilage or joint contusion, treatment must take account of ligamentous injury and the conditions that foster repair.

Inflammation is a process of reaction by living cells to some agent or condition inimical to their normal existence. This series of changes is properly recognized, therefore, as the first line of repair of actual or referred injury. Physiologic reaction from trauma witnesses, then, the phenomena of hyperemia, succeeded by stagnation and that, in turn, by exudation. Perivascular metabolism consists of what might appropriately be called "auto-debridement," characterized by the scavenger activities of the leucocytes, and the proliferation of fibroblasts into new connective tissue. The process of repair (anabolism) may be said to be launched when the field is cleared of debris.

Since ligaments have no blood supply and only

*Read before the American Academy of Physiotherapy, Boston, Mass., October 16, 1925.

that nutrition supplied by bathing in the juices exuded from other tissues, and since rest of the part slows the blood current, stagnates the resorption of lymph and therefore renews it in the tissues more slowly, it is plain, theoretically as well as in practice, that treatment to be most effective for prompt resolution of them should contemplate such use as will foster the nutrition indispensable for repair of the lacerated tissues and at the same time prevent further strain upon the affected ligaments.

The basic therapeutic wisdom upon which rests the preclusion of needless sequelae must provide more thoughtfully applied mechanical aids than are yet sufficiently often seen. If Thomas was right, if Bier's passive hyperemia is logical, if physiological repair is well conceived, we are constrained to have injured parts placed at ease with unimpeded circulation; to maintain equilibrium in muscles of opposition; to mobilize adjacent joints, the while protecting torn ligaments; to take disturbing strain and weight from damaged structures, in order to

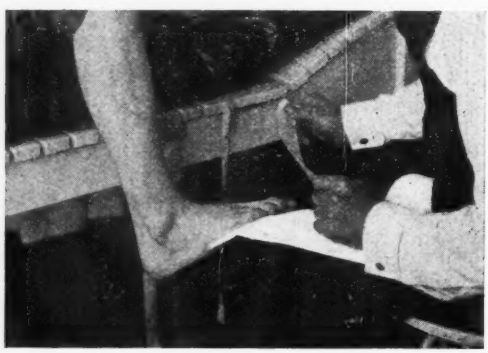


FIGURE 1. The prerequisite to efficiency of the dressing in the injury to the external lateral ligament is passive flexion of the foot to less than 90 degrees with slight eversion.

promote tissue regeneration, and to encourage the earliest exercises commensurate with the foregoing precepts.

There will be no dispute that when the joint is firmly held by support, the patient can, as a rule, walk with comfort; and he is encouraged to do so. For, functional use, provided it does not cause additional injury, is the most effective stimulant of repair; thus the patient, applying, as it were, an automatic massage, cures himself. May we not, then, take by assent that, as soon as the acute symptoms of a severe sprain subside and in every case of moderate pain, joints should be partially immobilized to maintain torn ligaments in approximation and to nurture, at the same time, the reparative processes by the remedial measures most conducive to recovery.

In 1902 the writer, then coaching an Eastern college eleven, had one of his strongest men laid out with a severe sprain two days before a most important game. By the time cold compresses had limited the acute inflammation, a form of strapping had been devised that allowed the man to play the next day and all other days. Continuously during the past twenty-three years, the experience with this form of strapping has been the same—not a single day's absence from play or daily duty; the repair process is prompt and subsequent sprain has never occurred with the foot in proper relation to the leg.

FIGURE 2. First strap is carried from the inside of the calf over the instep, around the cuboid bone under the sole and back to the dorsum of the foot for secure anchorage.

The essential of the author's method, illustrated by the accompanying slides, is the posture of the foot in relation to the lower leg. The patient is seated upon a table at such level that his depending foot hangs 6 to 8 inches lower than the knee of the operator, who sits facing him. With adhesive straps 1½ inches wide cut to length, the patient's foot is placed upon the operator's knee so that the ankle is dorsally

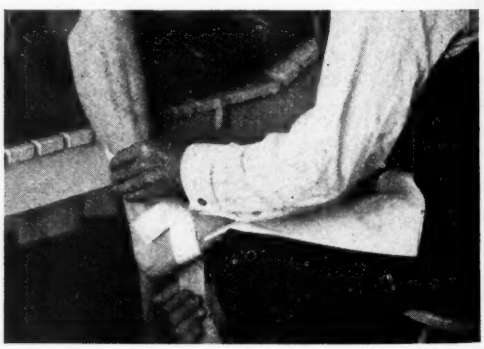


FIGURE 3. The corresponding strap starting on the outer side of the lower calf and passing over the scaphoid, the sole of the foot and returning over the instep. This strap balances the first, limiting eversion.

flexed to 80-85 degrees from the long axis of the tibia and rests the full weight of the extremity upon the head of the fifth metatarsal. The patient's leg must be wholly passive. This leaves the leg in straight alignment from hip to ankle joints and throws the foot into slight eversion.

The first strap runs spirally around the lower calf on the medial side, across the instep and cuboid, under the sole, falling into a natural sweep on the dorsum. This is the salient control of the hypermobility. The second strap is a

FIGURE 4. The third strap is a stirrup passing under the heel in the lateral plane of the tibio-astragloid joint.

FIGURE 5. A single circular strap without tension holds strap No. 3 in contact with the tissues in the isthmus above the malleolei.

counterpart in the opposite direction, acting to limit eversion and balancing the first. The third support is a stirrup from the middle of the calf and passes in the lateral plane of the ankle joint. The fourth and narrow strip retains the third support in contact with the leg at its isthmus above the malleolei. It is imperative that the patient's leg be maintained in the same posture throughout the dressing and his muscle action inhibited.

Walking is instituted at once with the foot carried in the four-square position—straight ahead. A little practice with shortened step and accelerated flexion of the knee at the completion of the stride obviates any necessity for a limp.

When perspiration permits the plaster to slip so that the angle between the foot and leg exceeds ninety degrees, the dressing must be reapplied. Sensitive skins require sedation and may necessitate periods of interruption from plaster. Otherwise no other treatment has been found necessary.

Since physiotherapy has dropped many of the scales from our eyes—or shall I say the scales from the eyes of many of us—it is recognized that diathermy, which has accomplished so much and is so full of wide promise, accelerates repair and abbreviates the dependence upon the plaster support. Daily removal of plaster is required for this treatment, preferably before retiring with reapplication in the morning.

Whatever of interest or merit this method may invoke, it is probably distinctive in having reached a maturity of twenty-three years' experience and durability before making a formal bow.

THE RELATIONSHIP OF THE WEATHER TO THE INCIDENCE OF PNEUMONIA IN THE CHILDREN'S HOSPITAL, BOSTON*

BY LEWIS WEBB HILL, M.D.†

THERE are periods each year, not necessarily during the winter months, when pneumonia in children is unusually prevalent, other periods when it is uncommon. Is it possible to correlate variations in the incidence of the disease with any definite weather conditions?

There are, of course, so many factors which determine the spread of pneumonia that it would be futile to assume that weather conditions alone are responsible for its increased or decreased frequency of occurrence. It might be possible, however, by a study of monthly weather conditions over a period of years, to indicate in a general way the parallelism of high or low incidence of pneumonia with various types of weather over corresponding periods. With this in view, the following study was undertaken‡.

*Read at the meeting of the New England Pediatric Society, April, 1927.

†From the Department of Pediatrics, Harvard Medical School, and the Medical Service, Children's Hospital.

‡I wish to thank Mr. Hathaway, of the Boston Weather Bureau, for the many courtesies he showed me, and for allowing me access to the beautifully kept records of the Bureau.

It is well known that Boston weather is extremely variable. Let us consider some of these variations.

HOURS OF SUNSHINE PER MONTH

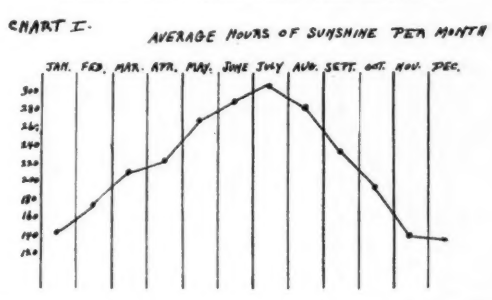
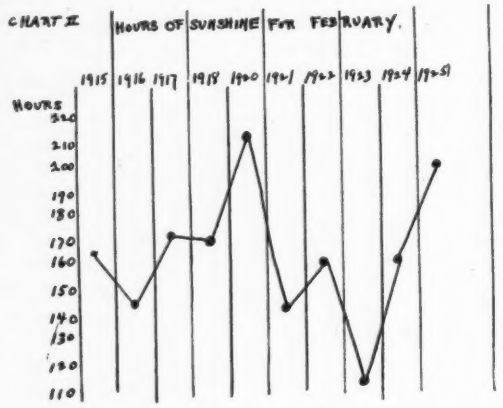


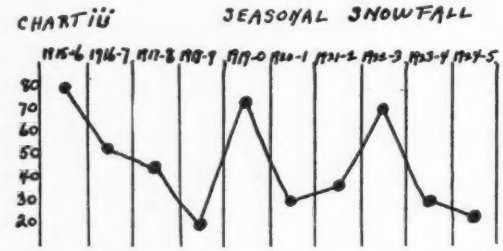
Chart 1 shows the steady rise of the monthly hours of sunshine to a peak of about 300 hours in July, and a rapid drop to a minimum of 136 hours in December. These are averages, taken over a long period of years. If a study of any one month is made, it is found that there are

tremendous variations from year to year. Chart 2 shows this for the month of February from 1915 to 1925 inclusive.



SNOWFALL

In this climate, the seasonal snowfall likewise shows great variations. Chart 3 indicates the seasonal snowfall over a period of eleven years.



The snowfall in any given month from year to year varies even more than the seasonal; in February, 1925, there was too small a trace to measure; in February, 1920, there was 32 inches; in March, 1924, 7 inches; in March, 1916, 33 inches.

RAINFALL

It is quite surprising to see that the average rainfall for each month is remarkably alike, if figures are taken over a long period of time. Table 4 is from the records of the last fifty years, and shows the average rainfall per month.

AVERAGE RAINFALL PER MONTH

January	3.59 in.	July	3.44 in.
February	3.29	August	3.75
March	3.82	September	3.15
April	3.52	October	3.33
May	3.26	November	3.75
June	3.05	December	3.33

Wide individual variations from these averages, of course, occur. In October, 1924, there was only .06 inch; in November, 1916, .59 inch; and in September, 1914, .21 inch. The other extremes for these months are 8.84 inch, 11.03 inch, and 11.95 inch.

TEMPERATURE

The mean temperature of any given month varies considerably from year to year, which is shown in Table 5. The daily variations are too well known to need comment.

TABLE 5

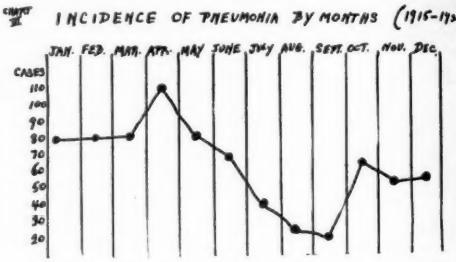
MEAN TEMPERATURE FOR FEBRUARY

1915	33.2	1919	32.6	1923	23.4
1916	25.5	1920	27.6	1924	26.6
1917	25.8	1921	32.6	1925	38.0
1918	26.9	1922	32.0		

From these few tables, it is at once apparent that there is a great deal of variation in the weather conditions of any given month from year to year.

Let us now turn to the monthly incidence of pneumonia in the Children's Hospital.

From 1915 to 1925 inclusive, there were 774 cases of pneumonia admitted. By far the greatest incidence was in April, the lowest in September, shown on Chart 6.



In Table 7, I have recorded all the months for eleven years when the admissions for pneumonia were usually high or unusually low for that particular month. There were 13 months out of the 132 under investigation when the number of admissions was decidedly above the average for the month, and 12 months when it was decidedly

TABLE 7

HIGH INCIDENCE	SUN	TPR.	SNOW	RAIN	LOW INCIDENCE	SUN	TPR.	SNOW	RAIN
JAN. 1915	LOW	HIGH	LOW	HIGH	JAN. 1920	NORM.	LOW	HIGH	NORM
JAN. 1917	LOW	HIGH	HIGH	NORM	JAN. 1921	HIGH	HIGH	LOW	LOW
JAN. 1919	LOW	HIGH	LOW	HIGH	JAN. 1922	HIGH	LOW	LOW	LOW
FEB. 1920	LOW	NORM	HIGH	HIGH	FEB. 1921	NORM	HIGH	HIGH	NORM
FEB. 1922	VERY LOW	HIGH	NORM	NORM	FEB. 1923	NORM	LOW	NORM	LOW
MAR. 1919	VERY LOW	HIGH	LOW	HIGH	MAR. 1923	LOW	LOW	NORM	LOW
MAR. 1920	NORM	NORM	HIGH	HIGH	APR. 1919	LOW	LOW	NORM	LOW
MAY 1916	NORM.	NORM		NORM	OCT. 1923	HIGH	NORM		NORM
MAY 1918	HIGH	VERY HIGH		LOW	OCT. 1924	HIGH	NORM		LOW
JUNE 1923	NORM.	HIGH		LOW	DEC. 1916	HIGH	NORM		NORM
JULY 1921	LOW	NORM		HIGH	DEC. 1922	LOW	NORM		NORM
OCT. 1918	LOW	NORM		LOW	DEC. 1925	NORM	NORM		NORM
DEC. 1915	NORM.	NORM		NORM					
	8 LOW	7 HIGH		6 HIGH		3 LOW	2 HIGH		1 HIGH
	1 HIGH	0 LOW		3 LOW		5 HIGH	5 LOW		6 LOW

below the average. It should be possible, by studying the weather conditions for these "high" and "low" months, to draw conclusions as to what sort of weather is favorable or unfavorable for the occurrence of pneumonia. A

"low" or a "high" sunshine, temperature, snow or rain, means that the figure shows marked variation one way or the other from the normal figure which one would expect for that particular month.

An analysis of the table shows the following:

Sunshine

In the 13 months when pneumonia was abnormally prevalent, there were 8 months when there was an abnormally small amount of sunshine, and only one month when there was an abnormally large amount. In the 12 months when there were unusually few pneumonia admissions, there were only 3 months with a low sunshine and 5 with a high.

Temperature

In the "high incidence" group of months, there were 7 that were abnormally warm, none abnormally cold. In the "low" group, there were only 2 months that were abnormally warm, and 5 that were abnormally cold.

Snowfall

The amount of snowfall seems to bear no particular relation to the incidence of pneumonia, which can be seen from a study of the table.

Rainfall

In the "high" group, there were 6 months when the amount of rainfall was excessive, and 3 when it was low. In the "low" group, there was only one month with a high rainfall, and 6 with a low.

SUMMARY

In a period of 132 consecutive months, there were 13 when pneumonia was unusually prevalent in the Children's Hospital wards. A large number of these months were unusually warm, with an abnormally high rainfall, and abnormally few hours of sunshine. There were 12 months when the incidence of pneumonia was unusually low. Many of these months showed abnormally high sunshine and abnormally low temperature and rainfall. Very few showed low sunshine or high temperature and rainfall.

CONCLUSIONS

It is reasonable to assume that the incidence of pneumonia in the Children's Hospital is a fairly accurate index of its prevalence in the community. If this is true, it may be concluded that abnormally warm, rainy weather with little sunshine favors its occurrence, and that abnormally cold, sunny, dry weather is unfavorable to it.

MEDICAL PROGRESS

PROGRESS IN NEUROLOGY

BY A. MYERSON, M.D.

EPILEPSY

THE subject of epilepsy is one of the ancient and important problems of medicine. It has been variously regarded, with the bulk of opinion divided up into several main camps. In the early phases of medicine, demoniac obsession played the most important role. Naturally enough, this passed out. Later on, largely due to the work of the Freudians, and especially Pierce Clark of New York, complexes became a sort of substitute for the demon. The epileptic seizure became a sort of escape of the complexes. Most medical men, however, have sought for an organic basis to the syndrome, because it has been well established that in various pathological conditions of the brain, such as tumor, neurosyphilis, arterial disease, and trauma, seizures called epileptiform occur. The term "epileptiform" really represents a dodging of the question, since the seizures in these cases are often not to be distinguished from any seizures in idiopathic epilepsy. The term "idiopathic" itself, in those cases for which no organic basis can be found, represents one of the inferiority reactions of medicine in which a term meaning really nothing is substituted for the term "unknown," which is a frank statement of ignorance. At present, the term "epilepsy of unknown origin" covers the status of most epilepsy.

Some groups of workers have for years insisted that the epileptic attack, especially in children, was a reflex disturbance due to gastrointestinal conditions. Such procedures as diet, the salt-free diet of a decade ago, diet to correct the constipation so commonly present, diet which avoided so-called irritating substances, such as spices, tea, coffee, candy, and the like, and even operations on the gastrointestinal tract to overcome the effects of absorption of toxic substances from the bowel were placed in the forefront of etiologic and therapeutic work in epilepsy. Bacteria were "discovered" which explained the disease. These bacteria, however, were found to be either non-existent or existent in normal individuals, and disappeared from the literature.

The following is a survey of some of the work appearing during 1926 on epilepsy. Most of the references I owe to the kindness of Dr. William G. Lennox of the Boston City Hospital.

The psychogenic factor which, as above stated,

Clark has brought into prominence is further elaborated by him. He regards the epileptic as a person who is of the oral and alal-erotic type, that is essentially narcisstic. (For the benefit of the unenlightened in Freudian matters, the narcisstic person is one whose worship is directed toward himself, and especially manifests itself by autoeroticism.) This narcism produces a rigidity and inelasticity of the person incompatible with normal living—hence the explosive fit. (Clark, L. P.—"Psychology of Essential Epilepsy"—*Journ. Nerv. and Ment. Dis.*, N. Y., Vol. 63, 575, June, 1926.)

The mechanism of the attack has received a good deal of attention. Foerster (*Zentralb. f. d. ges. Neurol. u. Psychiat.*, Oct. 15, 1926) states that before the attack, there is a vasoconstriction and anemia of the brain with distinct diminution in volume. Foerster has observed this at least a hundred times in patients who had convulsions on the operating table with the brains exposed. At the same time, he states, there is a rapid fall of cerebrospinal fluid pressure. This is followed by venous stasis with bluish-violet coloration of the surface and an increase in spinal fluid pressure. Foerster thus believes that there is much to say in favor of the vasomotor theory of epilepsy. In general, Foerster is an organicist in his conception of epilepsy, and lays the burden of its cause upon organic disease of the brain or on changes in the autonomic and sympathetic nervous systems.

Barnes ("Cerebral Manifestations of Paroxysmal Tachycardia"—*Am. J. of Med. Sciences*, Phila., Pa., Vol. 171, 469-624, April, 1926) reports cases from the Mayo Clinic in which epileptiform seizures occurred as a symptom of paroxysmal tachycardia, thus substantiating the relationship of epilepsy to circulatory disorder.

W. Spielmeyer (*Zentralb. f. d. ges. Neurol. u. Psychiat.*, Oct. 15, 1926) favors the circulatory theory, and also reports brain-lesions. The sites of lesions relating to epilepsy were the cerebellum and the cornu ammonis. He found these changes in 80% of 126 cases of idiopathic epilepsy. He believes that the changes in the brain are secondary to changes in the arteries.

Tilmann (*Deutsche med. Woch.*, Berlin, Vol. 52, p. 1595, Sept. 17, 1926) also states that he has found pathological changes in the cortex and meninges of the brain in his operative cases of epilepsy. The difficulty with such work as his is that cases which are operated for epilepsy are those in which brain changes are suspected, in which the convulsion is Jacksonian, and, therefore, there is a selection of material which naturally would show brain changes of one type or another. Furthermore, what one observes in operation cannot be placed in the field of pathological research, since microscopic examination is not made, and since the operation in itself contributes pathological appearances.

Spinal Fluid Studies in Epilepsy

W. G. Lennox (*J. Nerv. and Ment. Dis.*, Jan., 1926) in a report of 33 cases, states that almost all have normal spinal fluid pressures. In one patient, in whom spinal fluid pressure was very high during the severe convulsions, drainage was followed by a complete cessation of seizures for a period of eight hours.

H. A. Patterson and Paul Levi (*Arch. Neurol. and Psychiat.*, March, 1926) report that spinal fluid pressure is usually at the upper borderline of the normal, and that there is some increase during the attack. An unusually high percentage of positive reactions was encountered with the colloidal tests and the gold curve was that of the cerebrospinal syphilis type. (This is not the experience of the Boston City Hospital, Nerve Department.) In general, no very definite changes other than those above stated were found except that the sugar values were low, and that the ninhydrin reaction was positive in some cases.

Biochemical Examinations

Wuth (*Bull. Johns Hopkins Hosp.*, June, 1926) states that marked changes occur in the cellular and chemical blood constituents. During convulsions polynuclear leukocytosis and a diminution of the eosinophiles occurs. Non-protein nitrogen, uric acid, creatinin, and serum protein show an increase during, or shortly after convulsions. These changes are not specific and may be linked up with the high physical activity of the fit. Thus, Wuth's work is in harmony with the previous biochemical studies in being fundamentally negative in so far as a biochemical etiology of the attack is concerned.

The most interesting developments during the past year have been further contributions concerning the question of acidosis or alkalosis in epilepsy and the effect of acidbase changes on seizures. None of the work is conclusive in so far as the blood of the epileptic between attacks is concerned. Thus Frisch and Fried (*Zeitschr. f. d. ges. exp. med.*, Vol. 49, 462, 1926) measuring the pH of the serum of 23 patients, found measurements which, on the whole, were normal. Marrack and Thacker (*Brit. J. of Exper. Path.*, London, Vol. 7, p. 265, Oct., 1926) confirm this. Dautrebande (*Cpt. rend des séances de la soc. de biol.*, Vol. 94, pp. 133-135, 1926), who reached the conclusion that there may be an alkaline substance in the blood of epileptics which does not combine with carbon dioxide, has reached his conclusion without a sufficient number of cases.

It seems to be established that producing acidosis diminishes the number of attacks. This can be brought about by fasting or the use of the ketogenic diet, whereas conditions which produce alkalosis, such as the administration of bicarbonate and over-ventilation of the lungs, increase the seizures. (W. Jacobi—*Zeitschr. f. d. ges. Neurol. u. Psychiat.*, Vol. 102, 1926; Vollmer and Serebrijski—*Zeitschr. f. kinderh.*, Vol. 41, p. 314, June, 1926; Karger—*Klin. Wochenschr.*, Vol. 5, p. 502, 1926.)

Amongst those who report on the treatment of

epilepsy by the ketogenic diet are Talbot, Metcalf, and Moriarty. I refer the reader to the original article in the BOSTON MEDICAL AND SURGICAL JOURNAL, Vol. 196, No. 3, p. 89, Jan. 20, 1927, for details. They conclude that:

"I. The ketogenic diet fulfills the need of the child. Urine analysis indicates that one gram of protein per kilogram of expected body weight maintains nitrogen equilibrium.

"II. A marked ketosis may be maintained over a long period of time without any unfavorable symptoms. On the contrary, the general health of the patients has been excellent, which may in part be attributed to the regulation of their habits.

"III. A simplified method of calculation of ketogenic diets is employed, expressed by a ratio of the food constituents.

Total Caloric Requirements=9x[R(C+P)]+4C+4P.

"IV. The cases treated have been limited to that group of children having epileptiform convulsions of undetermined etiology.

"V. The improvement of the epileptic condition and the blood chemical changes in children produced by a ketogenic diet are similar to those produced by fast.

"VI. A diet sufficiently ketogenic to produce a marked ketonuria usually results in a definite improvement of epileptic symptoms."

Very little mention is nowadays made in the literature of the role of heredity in causing epilepsy. It may be stated as a general law in medicine that when little is known of a disease, much is ascribed to heredity, and that when much is known, little is ascribed to heredity. This has been the history in general paresis, tuberculosis, rickets, paralysis agitans, and many other diseases. The fact is that there are very few cases of epilepsy in which several members of the same family are involved compared to the tremendous incidence of epilepsy, and it is futile to link together as hereditary the epilepsy of a child and some peculiarity in temperament in a parent.

Despite the trend of present day researches indicating that the ketogenic diet is of value, one may stick to luminal as having value in lessening the number of attacks, and there is empirical importance in the regulation of the habits of the individual, especially in the care and treatment of constipation and in correcting marked eyestrain, bad posture, and such existing obvious defects.

MIGRAINE

Migraine received a good deal of attention during the past year. Bramwell (*Brit. M. J.*, Oct. 30, 1926) believes that there is a vascular basis, and that the paroxysm is probably a consequence of arterial spasm. He believes that the symptoms of the migraine attack, which range from scotoma to hemiplegia, hemianesthesia, and oculo-motor paralysis, are to be accounted for by local spasm and subsequent relaxation of meningeal or cerebral arteries, or of both. In the opinion of the reviewer, it is difficult to account for such marked symptoms as occur in many cases of migraine, symptoms which are indubitably organic, and for their rapid disappearance without bringing into play the circulation as a factor. MacMullin (*Brit. M. J.*, Oct. 30, 1926) cites cases substantiating this point of view. Holmes brings against this vascular theory the fact that vasomotor fibres are not found in the cerebral vessels.

Blitzen and Brams (*J. A. M. A.*, Mar. 6, 1926) point out that abdominal pain is often an outstanding symptom in the clinical picture. It is apparent that the gastrointestinal tract is markedly affected in migraine, as is evidenced by the common vomiting. Sometimes the disturbance hits structures lower than the stomach, and diarrhea and abdominal pain result.

Spinal Fluid Content

The spinal fluid sugar increase formerly reported as definitely found in encephalitis has not been well substantiated, and it is becoming the prevailing opinion that while there is an increase in many cases of encephalitis, one must not lay too much stress upon the sugar increase, except in differentiating encephalitis from purulent meningitis (W. P. Stowe—*J. Lab. and Clin. Med.*, Jan., 1926). Tuberculous meningitis gives values very definitely higher than the run of purulent meningitis, and definitely below other conditions which may be mistaken for it. In other words, a high sugar content rules out meningitis. A very low sugar content with meningeal symptoms indicates a purulent type, and as compared with most of the meningites, the tubercular form has a high sugar content.

Osnato and Killian (*Arch. Neurol. and Psychiat.*, June, 1926) report that there is an increase pari passu of the lactic acid of the spinal fluid with increase of the lactic acid of the blood. Spinal fluid obtained from the cases of meningitis show high figures for lactic acid.

PNEUMOCOCCUS MENINGITIS

A case is reported of recovery from pneumococcus meningitis. The case report leaves little doubt that this condition was actually present. The writers (H. L. Ratnoff and A. M. Litvak—*Arch. Pediat.*, July, 1926) attribute the recovery to the use of "optochin hydrochloride." It should be used together with antipneumococcus serum and given intraspinally. Five c.c. of a 1/1000 solution in salt solution per kilo of body weight is the dose recommended.

BRAIN TRAUMA

A. S. Maclair (*J. A. M. A.*, Mar. 6, 1926) formulates his experiences on brain trauma as follows:

"I. Skull fractures in themselves are not

fatal; it is the accompanying complications that render these cases so disastrous.

"2. Head injuries, if severe, have a more favorable prognosis if a linear fracture of the vault is present to assist, as far as possible, in the prevention of the formation of cerebral edema.

"3. Hematomas of the scalp overlying fractures should be evacuated to prevent infection.

"4. Intracranial hemorrhage should be drained either by repeated lumbar punctures or, if that fails, by cranial drainage.

"5. Cerebral edema is best treated by dehydration, with epsom salt by rectum, repeated lumbar punctures and subtemporal or suboccipital decompression as the occasion may demand.

"6. The spinal mercurial manometer and its interpretation are absolutely essential to determine the accurate degree of intracranial pressure.

"7. Operation is contraindicated in shock and medullary edema.

"8. Contusion and laceration of the brain are healed by nature.

"9. Osteomyelitic bone must be removed.

"10. Brain abscess must be drained through a clean field."

To the above, the reviewer subscribes, with this statement, that the water manometer of Ayer is far better than the spinal mercurial manometer, and much less expensive. It is to be emphasized that the state of consciousness, the pulse rate, the temperature, and the absence or presence of focal symptoms, constitute the best general guides in determining the condition of the patient, the prognosis, and the treatment indicated. The spinal puncture constitutes a remarkably accurate guide to the condition of the patient, if properly used, and, as emphasized by Donald Munro (Boston Med. and Surg. Jour., Dec. 24, 1925), is a valuable therapeutic measure, to be used in a more or less routine way in cases of head injury.

A. A. McConnell (*Irish J. M. Sc.*, Jan. 1, 1926) makes especial reference to the slow epidural, subdural, and subarachnoid hemorrhages after minor injury to the skull. These hemorrhages may not become clinically manifest for several days after the injury. When an individual who has received a head injury shows, even after the lapse of a week or more, disturbance in consciousness, drowsiness, perhaps slight paresis, Babinski sign, and some engorgement of the vessels of a disk, such a hemorrhage may be suspected, and the question of surgical intervention ought to be raised.

ENCEPHALITIS

Encephalitis continues to occupy a foremost place in the neurological literature. Nothing new has developed with respect to the infective agent. Most of the work which has been done to confirm or disprove the identity of the virus producing herpes and the infective agent of encephalitis has not tended to confirm the earlier observations of Levaditi and his followers. (F. Breinl—*Arch. Path. and Lab. Med.*, Sept., 1926; Zinsser and Fee-Fang Tang—*J. Exper. Med.*, July 1, 1926; F. Stern—*Zentralb. f. Bakt.*, Vol. 97, 1926.)

Rosenow's streptococcus theory has received some confirmation from the work of Evans and Freeman (*Pub. Health Rep.*, June 4, 1926). Their statement is that a pleomorphic streptococcus, highly virulent for rabbits when inoculated intracererally, was obtained from the nasal washings, heart blood, and mesencephalon of a case of epidemic encephalitis. The lesions occur in the central nervous system and there is a distinct resemblance to the inflammatory process found in encephalitis.

E. C. Rosenow (*Arch. Neurol. and Psychiat.*, June, 1926) believes that a streptococcus of similar type to the streptococcus which he described as causative of epidemic encephalitis is the causative agent in epidemic hiccough. The packing of the nose of an animal with gauze soaked in cultures brought about spasms of the diaphragm (rabbits, guinea pigs, and monkeys). The spasms of the diaphragm have been seen through an incision in the abdominal wall made under local anesthesia, and by means of the fluoroscope. During the severe spasm, audible hiccough was frequently noted in rabbits and monkeys. Rosenow names the organism "Streptococcus singultis." Those animals that succumb show symptoms of encephalitis. Rosenow believes that the changes in the localizing power of the streptococcus, that is, change in symptoms from those of hiccough to those of myelo-encephalitis, are due to cultural conditions, passage through other animals, and the fluctuations in the characteristics of the streptococcus thus produced. His discussion of the use of serums and vaccine treatment cites striking results, but in too few cases to be of definite value.

That encephalitis becomes a chronic infective disease, and that the so-called sequelae are to be classed in the same group as the symptoms of general paresis in relation to syphilis, namely that they are due to persistent presence of the infective agent, is the conclusion of Walter Freeman (*J. A. M. A.*, Nov. 13, 1926). This view is, on the whole, accepted by most of the investigators, and any treatment of these sequelae, such as the postencephalitic Parkinsonian disease, needs to take this into account.

There is a very large literature, which the reviewer will not cite in detail, concerning the so-called residuals of encephalitis. It seems to be undoubted that following encephalitis, mental disease, narcolepsy, eye muscle changes, polydipsia, and character alterations have appeared, and the future history of a case of a patient who has had encephalitis is a matter for great concern and close observation.

The treatment of the nervous system, especially the Parkinsonian group of diseases following encephalitis, has not been satisfactory. Mercurochrome is declared by Hengstler (*Minnesota Med.*, May, 1926) of no value. Swedish investigators (Marcus, Kline and Höglund—(*Svenska Läkartid.*, Vol. 23, 1926) are using inoculations of Treponema Duttoni of African relapsing fever. Katona (*Gyogyaszat*, Vol. 66, 1926—ab. in *Zeitschr. f. d. ges. Neurol. u. Psychiat.*, Sept. 1, 1926) reports fairly good results from the intravenous injection of sodium cacodylate, 50% solution given in increasing doses from 0.5 to 2 c.c. The total amount of the drug given was from 50 to 80 gms.

Hall (*Brit. M. J.*, Jan. 23, 1926), using large doses of tincture of belladonna, found that general improvement occurred with the use of 20 to 30 minims three times a day.

Other treatments recommended in the literature of the past year consist of autoserum treatment by Finlayson and Karnosh (*Ohio State M. J.*, April, 1926) and nicotin by Henry Moll (*Brit. M. J.*, June 26, 1926). One is not impressed that these are methods of treatment which offer any great degree of hope.

ROENTGEN TREATMENT OF BRAIN TUMOR

Roentgen treatment of brain tumor has been especially advocated for pituitary neoplasm. The more common anterior lobe adenoma which enlarges and erodes the sella, responds fairly well to roentgen-ray treatment. E. B. Towne (*Arch. Neurol. and Psychiat.*, Jan., 1926) believes that "all adenomas of the pituitary gland should be treated with moderate doses of roentgen ray, and progress should be followed by repeated perimetric examinations. Operation should be undertaken only if visual acuity and the fields do not improve. There is good reason to believe that when roentgen-ray treatment gives a favorable result, the risk is less and the result more lasting than that of surgical treatment."

F. C. Grant (*Atlantic M. J.*, April, 1926), who writes from Frazier's clinic in Philadelphia, believes that every operative procedure should be followed by x-ray applications, and that in the early cases where vision is not greatly interfered with, and where the sellar deformities are not extreme, x-ray therapy should be given a thorough trial before resorting to surgical measures.

NEUROSYPHILIS

General Paresis

It is the opinion of Plaut (*Zeitschr. f. d. ges. Neurol. u. Psychiat.*, Vol. 101, 1926) and Wilmanns (*Zeitschr. f. d. ges. Neurol. u. Psychiat.*, Vol. 101, 1926) that the early anti-syphilitic treatment is responsible for the incidence of neurosyphilis later on. Energetic and early treatment prevents the secondary reactions, but favors the type of syphilis found in civilized countries, namely parenchymatous neuro-syphilis. The biological properties of the spirochetes are changed by the action of chemicals, and these changed characters are to some extent inherited (by the spirochetes). Hauptmann (*Zeitschr. f. d. ges. Neurol. u. Psychiat.*, Vol. 102, 1926) goes so far as to state that no syphilitic should be prevented from having cutaneous manifestations during the second stage unless it is possible to kill all spirochetes during the primary stage.

The acetic anhydride-sulphuric acid test for general paresis was described by O. H. Baltz in 1923 (*Am. J. Psychiat.*, July, 1923). Harris (*Brit. M. J.*, Jan. 23, 1926) now describes the technic as follows:

"To one c.c. of cerebrospinal fluid in a small glass test tube add 0.3 c.c. of acetic anhydride. Shake the mixture well and then carefully add, drop by drop, 0.8 c.c. of concentrated sulphuric acid. The test tube is then held up against a white background and the presence of a lilac tint indicates a positive reaction; a brown-yellow or a red-yellow color is noted if the reaction is a negative one. The lilac color, characteristic of the positive reaction, may appear immediately after the addition of the sulphuric acid and disappear in a minute or so. Hence it is important to watch the cerebrospinal fluid closely as soon as the sulphuric acid is added, or a slightly positive case may be missed. In the majority of cases the lilac tint remains for at least five minutes."

The conclusions of Harris on this test are as follows:

1. The acetic anhydride-sulphuric acid test on the cerebrospinal fluid of cases of general paresis is positive in almost every case (97%) and hence is almost as reliable as the Wassermann reaction in this condition.
2. It is negative in almost every other type of mental disorder except certain cases of neurosyphilis (other than general paralysis of the insane).
3. It is a test which can be performed with great ease and rapidity and hence is of definite value to the clinician.

TREATMENTS

Malarial Treatment of General Paresis

Bunker and Kirby (*Arch. Neurol. and Psychiat.*, Aug., 1926) make a further report on their experience with the malarial treatment. Of 106 patients treated since September, 1923, 22 have died, and in 11 of these the malaria is held to be in part responsible for the fatal termination. Twenty-six are classed as unimproved; 8 are classed as slightly improved; 13 have been moderately improved; and 37 have achieved full remissions. The most favorable cases were the so-called manic type, the cases with expansive delusions were next, and the dementing types were the worst so far as remission was concerned. The Mayo Clinic reports, of a very small number of patients, that 25% showed a complete remis-

sion, and 35% were improved. They believe that arsphenamin should not be used for several months after the malarial treatment (O'Leary, Goeckerman and Parker—*Arch. Dermat. and Syph.*, Mar. and Nov., 1926).

Driver, Gammel and Karnosh (*J. A. M. A.*, Nov. 27, 1926), putting together the reports from twelve countries comprising 2,336 cases treated by malaria, find that 641 had complete remissions, and 616 incomplete remissions.

Nonne (*Med. Klin.*, Dec. 4, 1925) is still as optimistic as ever in his view that general paresis may be very definitely helped by the malarial treatment.

Other Infections Used in Treatment of General Paresis

Relapsing fever has been used in the treatment of general paresis by R. Schröter (*Zeitschr. f. d. ges. Neurol. u. Psychiat.*, Vol. 103, 1926); 47% of 140 cases had complete remissions; about 10% had partial remissions.

Plaut (*Internat. Clin.*, 36th Series, Vol. 1, 1926) has used spirochete Duttoni of African relapsing fever. He reports about as good results as have been cited by the workers with malaria.

Solomon, Berk, Theiler and Clay (*Arch. Int. Med.*, Sept. 15, 1926) have used rat bite fever, with results that are promising. The advantage claimed for this method is that the inoculation is less dangerous.

The rationale of all kinds of fever therapy in the treatment of general paresis depends, according to Schamberg and Rule (*Arch. Dermat. and Syph.*, Sept., 1926), on the fact that the spirochete pallida is biologically damaged or destroyed at a temperature far lower than that needed to injure other micro-organisms.

Reese of the Wisconsin Psychiatric Institute (*Wis. M. J.*, June, 1926) claims that on the whole tryparsamide is better than malarial treatment in that remissions are more constant and there is less danger.

MULTIPLE SCLEROSIS

The vogue of fever treatment has extended to the treatment of multiple sclerosis. Dreyfus and Hanan (*Deutsche med. Wchnschr.*, Feb. 26, 1926) used it on a few cases and claim improvement with the use. The fever was induced by the use of typhoid vaccine.

LIPIODOL

Lipiodol has completely substantiated the claims of its promulgators as a valuable aid in the diagnosis of cord tumors. Though many articles have been written on the subject, there is, on the whole, such complete agreement as to obviate the necessity of citing any one of them. That there is some degree of pain following this method of diagnosis and that there is meningeal irritation of an aseptic type, are drawbacks which do not outweigh the advantages.

SCIATICA

J. P. Martin (*Lancet*, May 22, 1926) gives an excellent account of the symptomatology and treatment of sciatica. He points out, first, that in sciatica the inflamed nerve is always tender; and, second, since the sciatic nerve sends no branches to the front of the thigh, any condition in which the pain extends to the front of the thigh or groin is not sciatica alone; third, sciatica causes no muscular paralysis and no gross sensory loss; and fourth, it may abolish the ankle jerk (it usually does), but never the knee jerk. He gives a long account of the treatment of this condition. He favors anodal galvanism as the method of choice for mild or moderate cases. He thinks diathermy is suitable for this same kind of case. He advocates the injection of oxygen for acute and subacute cases. The technique of this method of treatment is too long to be cited in this review, and the interested reader is referred to the original article. He advocates saline injection into the buttock in cases which have persisted for a month or six weeks. The object of this is to distend the sheath of the nerve, to relieve pressure, and to break down adhesions. This is a very valuable article, and should be read in the original.

SYMPATHETIC NERVOUS SYSTEM

There is a decided schism between the physiologists and the surgeons in regard to the sypmpathetic nervous system and its relationship to muscle tone. In previous reviews, it has been stated that Royle and Hunter claimed that one of the phases of muscle tone, the so-called plastic phase, was due to separate sympathetic innervation, and that by section of the sympathetic roots, excessive tone or spasticity disappeared. The physiologists and the anatomists are distinctly skeptical, if not contradictory of this finding.

Forbes and Cobb (*J. A. M. A.*, June 19, 1926) completely disagree, on the basis of their experiments, with the point of view of Royle and Hunter. They state that muscle responds on the all-or-none principle; further, that there is no contraction of the muscle in response to artificial stimulation of sympathetic nerve fibres, and drugs known to affect the sympathetic have no effect on the contraction of muscles. Their crowning argument is that surgical procedures carried on by other investigators do not give the results that Hunter and Royle obtained.

Davis and Kanavel (*J. A. M. A.*, June 19, 1926), who have operated on various diseases of the central nervous system, did not get results like those of Hunter and Royle. They concede that it is possible that removal of the sympathetic influence may, by chemical means, alter the contractility of muscles. They do not find any direct influence of the sympathetic upon muscle.

Asher (*Schweiz. med. Wochnschr.*, June 5,

1926), whose previous work seems to indicate that there was sympathetic influence on the tonus of muscle, now states that with improved technique, so that the spinal motor fibres are not injured, there is no evidence that extirpating of sympathetic produces changes in muscles. However, his attitude is that sympathetic innervation is possible, and that sympathetic influence on muscle tone should be further tested out.

Coman (*Bull. Johns Hopkins Hosp.*, March, 1926) is in accord with Forbes and Cobb that there is no direct relation between the sympathetic nervous system and the maintenance of posture or tonus of a limb.

Sympathectomy in Vasomotor Diseases

Davis and Kanaval (*Surg. Gynec. Obst.*, June, 1926) state that great relief of symptoms occurs when sympathectomy is done in Raynaud's Disease. They present the following conclusions:

"1. The peripheral blood vessels receive their sympathetic innervation in a segmental manner by way of the somatic nerves.

"2. Removal of the sympathetic innervation to an extremity in man is followed by hyperemia and hyperthermia which gradually disappear within one to two weeks following operation.

"3. In certain vascular diseases of the extremities characterized by paroxysmal vasomotor symptoms and the absence of organic vessel pathology, removal of the sympathetic innervation is followed by an improvement in symptoms. Among this group we may include erythromelalgia and Raynaud's Disease.

"4. There are many physiological factors concerned in the control of the peripheral circulation, of which the vessel musculature and caliber are but a part.

"5. In the present state of our knowledge concerning the pathology of the group of vascular diseases, known as vasomotor neuroses, we are unable to explain completely the effects produced by removal of the sympathetic innervation to the extremity."

Brown (*J. A. M. A.*, Aug. 7, 1926) gives a report of three cases from the Mayo Clinic in which, with Raynaud's Disease of the lower extremities, the extirpation of the second, third, and fourth lumbar ganglions and the sympathetic trunk and the stripping of perivascular tissue from the common iliac arteries brought marked clinical improvement which has persisted.

A good deal of discussion concerning periarterial sympathetic stripping in angina pectoris has appeared in the recent literature. Ranson, who is one of the leading anatomists, (*J. A. M. A.*, June 19, 1926) finds no anatomical background for the relief of angina pectoris by extirpating the left superior cervical ganglia, since it does not appear from anatomical researches that the afferent fibres from the heart are interfered with in this operation. Nor does Ranson find any anatomical reason why periarterial sympathectomy for the relief of angina pectoris gives good results, since the vasoconstrictive fibres do not follow the main arterial trunk into the limbs, but instead follow the spinal nerves into the subcutaneous tissue where they are given off for the innervation of the small arteries and the arterioles. There is a hiatus between the anatomical knowledge we have of the sympathetic nervous system and the reported clinical results of superior cervical sympathectomy and periarterial stripping which Ranson is not able to bridge.

CANCER AND THE ANSWER

Among the answers the State Department of Health has issued the following references to the clinics:

THE PROGRAM

The Massachusetts Department of Public Health is now launched upon a campaign for cancer control, which includes:

Clinics in several of the larger centers of the State. These will bring the best of modern diagnostic facilities within reach of all.

Speakers, all of whom are physicians, are ready to meet any group or organization which desires to consider the subject of cancer control.

Literature for distribution.

Hospital care for 90 cases of all types of cancer at the Pondville Hospital in Norfolk to supplement local hospital resources.

All service except hospital care is without charge.

LOCATIONS OF THE CLINICS

Lowell—Lowell General Hospital every Friday at 10 a. m.

Lynn—Lynn Hospital every Friday at 10 a. m.

Newton—Newton Hospital every second and fourth Fridays at 3 p. m.

Springfield—
Mercy Hospital, 4 to 5 p. m., June 24th.
Springfield Hospital, 4 to 5 p. m., July 1st.
Wesson Hospital, 4 to 5 p. m., July 8th.
Weekly, in regular rotation

Worcester—
Louis Pasteur Hospital, Mondays, 9 a. m.
Fairlawn Hospital, Tuesdays, 9 a. m.
Memorial Hospital, Tuesdays, 11 a. m.
City Hospital, Wednesdays, 9 a. m.
St. Vincent's Hospital, Thursdays, 9 a. m.
Harvard Hospital, Fridays, 10 a. m.
Hahnemann Hospital, Saturdays, 9 a. m.
Memorial Hospital, Saturdays, 11 a. m.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

DR. OSCAR RICHARDSON RETIRES

WITH this issue the services of Dr. Oscar Richardson as a regular participant in our coöperative undertaking—this series of case records—comes to an end on account of his retirement from his position as assistant pathologist at the Massachusetts General Hospital. He has put his intelligence and his patient conscientious observations into the study of these cases since we began discussing them in 1905, ten years before the publication of these Case Records was begun.

His unique service has been based on the fact that he has done the necropsies himself and with essentially the same plan of search, measurement and record throughout the whole period covered by this Case Records series and for many years before that. He has not entrusted the work to others and he has not hurried through the "uninteresting" cases, but has looked for everything and put down everything relevant in every case, so far as this is humanly possible. The personal equation has been the same throughout.

As a result of this when one asks for any particular weight, measurement or relation from the standpoint of some tabulation or research one almost always finds that in his conscientious routine he has kept the all-important record of what one wants.

His other outstanding virtue for work of this sort has been his ability to tell essentials from nonessentials and to put his results into plain words. Thus in our teaching exercises his contribution has always been vital and clear.

We regret sincerely that we cannot have him as a regular member of our group henceforth, though we hope for occasional assistance from him still.

RICHARD C. CABOT.

CASE 13291

DYSPNEA AND MELENA

MEDICAL DEPARTMENT

A German-American janitor fifty-four years old entered January 10 complaining of dyspnea of ten days' duration.

December 30 after running upstairs he became very dyspneic, felt his heart pound and noticed a pressure pain or feeling of heaviness over the precordia. His vision was blurred. He soon recovered from the attack. Three days later he shovelled snow and "caught a slight cold." Again he was very dyspneic, had palpitation and a feeling of precordial pressure. His doctor put him to bed, where he had to sit propped up with pillows, and gave him several hypodermics and some white pills which did not relieve him but nauseated him and made him vomit. While in bed he frequently had attacks of palpitation, especially on moving about or twisting. In the past few weeks he had lost some strength. In three weeks he had lost eleven pounds. For two weeks his appetite had been poor. Three days before admission he had sharp shooting pains in the calves of his legs, not recurring, and his lower legs and ankles were somewhat swollen. He had been digitalized.

His mother died of heart trouble.

He had tonsillitis in childhood. At sixteen he had gonorrhea. He denied syphilis. He had had a "cigarette cough" for forty years, often with a little sputum.

In the course of his work he did much stair climbing.

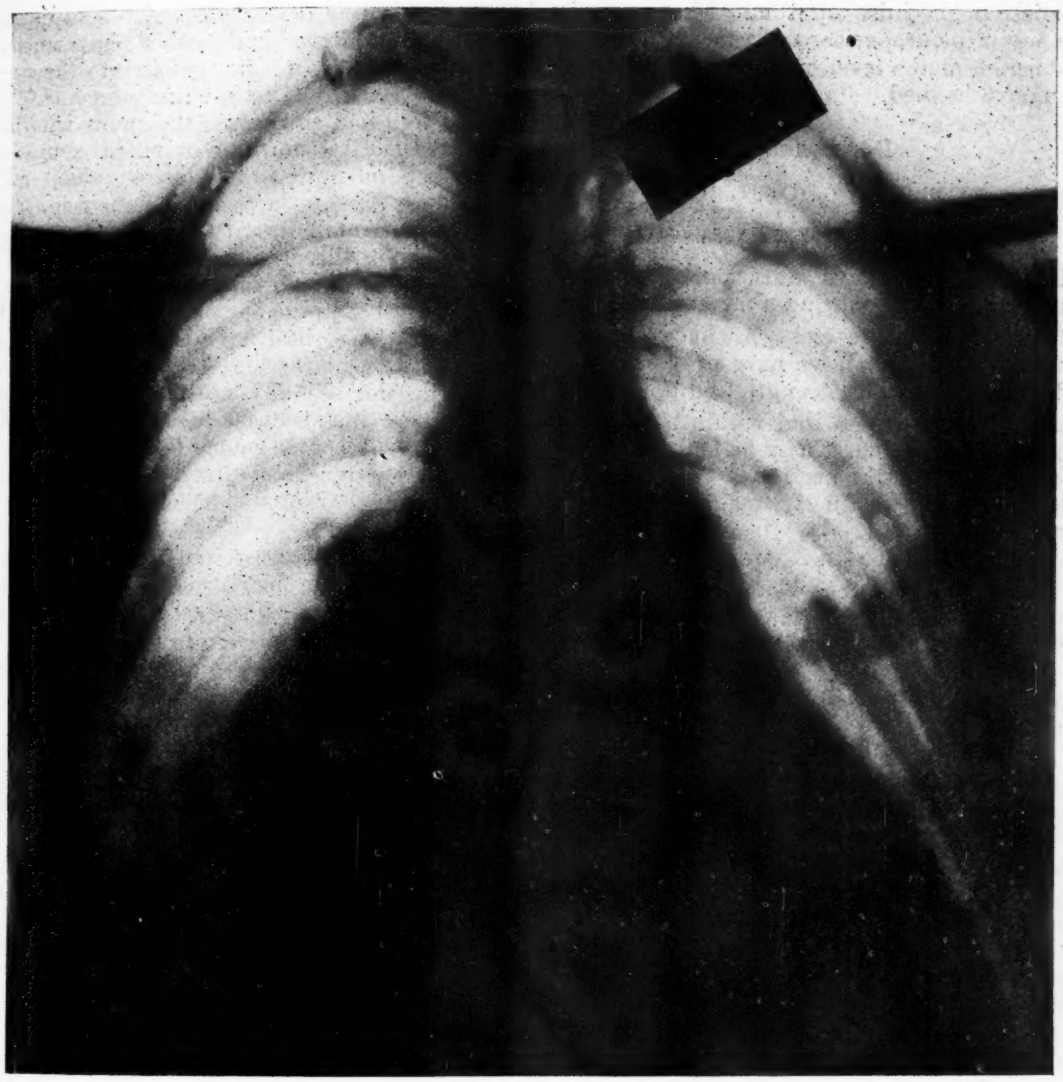
Clinical examination showed a fairly well developed, nervous man lying quietly in bed, occasionally coughing and breathing with some difficulty in paroxysms. The sclerae were slightly icteric, the skin brown (icterus), the lips dry and wrinkled. The trunk and legs showed a papular and linear red rash with many excoriations. Teeth carious. Pyorrhea. On the upper left faucial pillar and to a less extent on the right were several irregular white patches of exudate with diffuse redness of the throat. Cervical, axillary and inguinal adenopathy. The apex impulse of the heart was 10 centimeters from midsternum in the fifth space, coinciding with the left border of dullness 2.5 centimeters outside the midclavicular line. The right border was 1 centimeter from midsternum, the supracardiac dullness 7 centimeters. A systolic thrill at the apex. A systolic murmur over the precordium. An early high-pitched diastolic murmur, best heard at the second and third spaces to the left of the sternum. An early mid-diastolic heard at the mitral area. Typical Corrigan pulse. Blood pressure 200/60 to 250/55 to 150/10. Electrocardiogram: normal rhythm, rate 80, slightly diphasic T_2. Fine crackles at the base of the left lung. Abdomen held very stiff. Edema of both feet. Small external hemorrhoids. Fundi negative. Pupils reacted slightly, otherwise normal. Knee-jerks normal.

Urine normal in amount, yellow-red or pink at two of five examinations, cloudy at two, specific gravity 1.022 to 1.030, the slightest possible trace to a very slight trace of albumin twice, bile present twice, a questionable trace of sugar once, very rare leucocytes once. Renal function 45 per cent. Blood: 10,750 to 23,750 leucocytes, hemoglobin 95 to 50 per cent., reds 4,376,000 to 2,024,000, slight anisocytosis, no poikilocytosis

or stippling, reticulated cells two to five per cent. Two Wassermanns and a Kahn test negative. Non-protein nitrogen 72 to 76 to 28 per cent. Icterus index 15 to 20 January 13, 20 to 25 January 18, 5 February 1. Two throat cultures negative. Dark field of throat lesion negative. Vital capacity 1700 to 2100. Coagulation time 7½ to 11½ minutes. Bleeding time 5 minutes and 30 seconds. Stools: guaiac strongly positive in five of seven examinations, macroscopic blood twice, bile once. Sputum, by Smith stain: many leucocytes, a moderate number of encapsulated Gram-positive diplococci, some intracellular.

X-ray January 17 showed the heart shadow definitely enlarged. The increase was largely downward and to the left. The supracardiac dullness was also increased. There was a localized area of dullness at the right base which occupied the region of the right lower lobe.

Consultations. Laryngologist. Evidently a left recurrent paralysis. *Dermatologist.* I believe that the slight amount of jaundice is probably responsible for the pruritus, which again is responsible for most of the skin lesions. *Cardiologist.* . . . Well marked Austin Flint murmur at the apex. *Psychiatrist.* Very marked arteriosclerosis . . . but no dementia in marked degree. Peripheral arteriosclerosis very marked. Retinal arteriosclerosis in moderate degree only.



The heart shadow is definitely enlarged. The increase is largely downward and to the left. The supracardiac dullness is also increased. There is a localized area of dullness at the right base which occupies the region of the right lower lobe.

The patient required an occasional injection of morphia, threatening to jump through the window if he did not have it. He had anginal attacks some ten times a day. It was thought some of these might be feigned. He said nitroglycerin relieved the attacks but "made him nervous," so that he preferred morphia. He was given luminal and paraldehyde by routine and sterile hypodermics when he urgently demanded morphia. The icterus continued.

January 23 he passed more than a pint of

what seemed to be nearly pure, slightly changed blood by rectum. The blood pressure dropped to 120/90. He was quiet under morphia, with a pulse of 80. Following this the orthopnea seemed much improved. There was marked anemia, but the pulse tension gradually increased. After the anemia set in he was much quieter. January 31 the stools still gave a positive guaiac test. February 1 he was coughing up thick prune-juice sputum. The next day there was a pleuropericardial rub at the left of the sternum at the level of the third interspace. February 3 he died.

Discussion

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

"Dyspnea of ten days' duration." That is a pretty short period to bring a man to a hospital.

"In the past few weeks he had lost some strength." That is all we have previous to the ten days—some loss of strength and appetite.

I guess he was digitalized within a few days, not a very long period. Why he had those pains in his legs I do not know. It is not a symptom of anything I can distinctly imagine to be present in this case.

I do not recognize any such thing as "cigarette cough." It is true that people cough a little more when they smoke. I do not know any cough peculiar to cigarettes. So it seems to me that we have to think of some other factor, a factor of unusual sensitiveness, as well as the irritant.

What have we a right to expect in the physical examination on the basis of all this? It seems to me an arteriosclerotic type of heart trouble. The only type that is inheritable is that dependent upon vascular trouble. You have a suggestion there in his family history. The extreme acuteness of this suggests a vascular trouble, very possibly a coronary artery trouble. It seems to me that disease in the heart wall is a thing that should be first in our minds, second a chronic hypertension, of which I see on looking down the record he seems to have some evidence, though it may easily be something else.

NOTES ON THE PHYSICAL EXAMINATION

"On the upper left faucial pillar and to a less extent on the right were several irregular white patches of exudate with diffuse redness of the throat." The sort of thing to make one take a culture for diphtheria, but it probably is not that.

"A systolic thrill at the apex." That is very important if true.

Typical Corrigan pulse means the sort of thing you never get except in aortic regurgitation. If that blood pressure is correct we can take the one datum as supporting the other.

The description of the urine makes us think of passive congestion. Certainly no evidence pointing to the kidneys.

"Reds 4,376,000 to 2,024,000." All in two weeks! Either he had a hemorrhage or someone counted wrong. That is an extraordinary change if true.

Now we very suddenly come upon a consultation of the laryngologist, evidently something being omitted from the record. He tells us that the left vocal cord is paralyzed. I suppose the patient must have had either hoarseness or aphonia, but they did not get it into the record.

"Well marked Austin-Flint murmur." We might have had to discuss the point whether it was Austin-Flint murmur or mitral stenosis.

There is no word in the history about mental symptoms, or why they called the psychiatrist.

"The patient required an occasional injection of morphine, threatening to jump through the window if he did not have it." I do not know what for. I suppose for pain connected with an aneurysm. We shall have to discuss that later. At any rate that is where the psychiatrist comes in. I do not see any reason for consulting a psychiatrist just because a man wants morphia for pain. Of course morphia relieves angina a great deal better than nitroglycerin, and a good deal longer. I do not blame him at all for preferring morphia.

"January 23 he passed more than a pint of what seemed to be nearly pure slightly changed blood by rectum." That is why his red count went down so.

This X-ray is not a seven-foot plate, so we can not tell much about the size of the heart. But I have no doubt that it is enlarged. There is congestion at the right base, fluid I suppose. There is nothing there that I can see to give any support to the idea of aneurysm in the chest.

The X-ray interpretation sounds as if they thought it was pneumonia and not fluid.

DIFFERENTIAL DIAGNOSIS

The best evidence we have is of aortic regurgitation. We have three striking signs of that, the murmur, the pulse and the blood pressure.

A Student: How about that increase in the diastolic blood pressure after the hemorrhage?

Dr. Cabot: I do not know what to say about it. It does not fit with anything that I can think of.

A Student: Would a relative insufficiency give it?

Dr. Cabot: I should not suppose so.

A Physician: The blood pressures were taken almost every day and they all showed a pulse pressure of one hundred or more. I do not know whether the blood pressure toward the end had gone back to the same type. The blood pressure on the day of death for instance is 180 systolic and nothing they could record diastolic.

Dr. Cabot: I think there must be some mistake about the 120/90. I think we can throw that out and start with what we first thought of,

aortic regurgitation. Why should he have aortic regurgitation? With a man of this age with no previous history or symptoms you ordinarily say syphilis. Against that we have nothing except two negative Wassermanns and a Kahn test. Eighty per cent. of syphilitic patients show positive Wassermanns. That is quite a staggering blow to that diagnosis. There are rare cases of aortic regurgitation not explainable either by syphilis or rheumatic endocarditis of the aortic valve, but purely by arteriosclerosis.

Now coming back to the cause of this aortic regurgitation, there is no reason to make us think it is rheumatic, and some evidence that it is syphilitic. I do not see how we can say it is not arteriosclerosis, although that is a rare cause. It is believed by most writers to be a cause of organic disease of the aortic valve.

A STUDENT: Don't you have to account for the recurrens paralysis?

DR. CABOT: Yes, and I do not know how to. There is nothing in the record about hoarseness or loss of voice, is there? Of course that is ordinarily due to an aneurysm pressing on the recurrent laryngeal nerve. There is nothing the X-ray men are more keen about than finding aneurysm. Since they did not find it I think we can safely say it is not there.

MISS PAINTER: The consultant wrote. "The left cord is in adduction. The right cord hyperactive."

DR. CABOT: I suppose that is evidence of recurrens paralysis, but I do not know how to explain it.

The next outstanding fact is this big hemorrhage that brought the red count down to two million. What the source of that is I do not know. I do not see how it can be an aneurysm, with no other symptoms or signs pointing in that direction. So far as I know it perfectly well can be a silent peptic ulcer or a cirrhosis of the liver, although cirrhosis of the liver would give us the vomiting of blood and not the passing down, because the dilated, fragile veins of the gullet are so high up. It seems a little more likely to be an ulcer independent of aortic regurgitation.

A STUDENT: Would it not be more changed if it came from so high up?

DR. CABOT: Yes, I should say so.

A STUDENT: Isn't it more reasonable to suppose that the bleeding was due to hemorrhoids?

DR. CABOT: I never heard of anyone bleeding that much from hemorrhoids. I am ready to vote against hemorrhoids as a possible cause of this bleeding.

A STUDENT: How about carcinoma?

DR. CABOT: I never heard it keep so quiet, that is, present so few symptoms as here recorded. I am against it.

A STUDENT: How about tuberculosis of the rectum?

DR. CABOT: I never heard of it so low down.

A STUDENT: Wouldn't you get more gastric symptoms with ulcer?

DR. CABOT: Usually, but some cases are absolutely silent, and perforation and hemorrhage is the first thing we know.

A STUDENT: How about the enlarged lymph glands? Is there any way to account for them?

DR. CABOT: Not that I know. Syphilis is a good thing to say.

A STUDENT: How about Hodgkin's disease?

DR. CABOT: What have you for evidence?

A STUDENT: The glandular enlargement, and perhaps a recurrens paralysis.

DR. CABOT: It ought to show in this X-ray.

A STUDENT: Don't you think that shadow is a gland?

DR. CABOT: I am resting pretty heavily on the radiologist. He does not think so.

A STUDENT: The anemia would go with Hodgkin's.

DR. CABOT: Yes, if one did not have the hemorrhage to account for it it would very well.

Adenopathy of course does not necessarily mean anything of importance. So that I do not see how we can put any weight on that.

DR. MALLORY: I wonder if the record has any more precise description of that pain for which he is asking morphia?

DR. CABOT: It is referred to as angina and not in my record referred to in any other way. That would go perfectly well with a diagnosis of arteriosclerosis or syphilis. I cannot rule out syphilis of the aorta merely from the absent Wassermanns.

A STUDENT: Isn't pain a feature of aortic regurgitation?

DR. CABOT: No, it does not cause pain. The things that go with it sometimes cause pain.

A STUDENT: Should we not be inclined to place much less evidence on the Wassermann and the Kahn and put more on the syphilis with the history of exposure and gonorrheal infection, and then the large supracardiac dullness and the adenopathy?

DR. CABOT: Yes. I certainly can not rule out syphilis. I do not think the enlarged supracardiac dullness, in view of that X-ray, means much. But certainly Wassermann and Kahn tests do not rule out syphilis.

I think I have gone as far as I can. I feel confident that it is aortic regurgitation of an organic type. I do not know whether it is due to syphilis, as suggested by the adenopathy, by the recurrens paralysis, or to arteriosclerosis. Either one of those two things would account both for the pain that he had and for the cardiac condition. Neither of them would account for the blood he passed by rectum, and as far as I can see we have to make some perfectly separate diagnosis such as peptic ulcer to account for that.

A STUDENT: How about his temperature at the end?

MISS PAINTER: The day before he died it was

103.4°. The day he died it was 102°. It was high throughout.

DR. CABOT: He had a high temperature for a good while at the end. It seems to me pneumonia is a good guess.

A STUDENT: How about portal thrombosis, to account for the hemorrhage?

DR. CABOT: Portal thrombosis independent of anything else that we know?

A STUDENT: In association with the arteriosclerosis, general vascular weakness.

DR. CABOT: Portal thrombosis gives rise to a good many evidences of portal obstruction, such as marked ascites.

A STUDENT: He had a board-like abdomen.

DR. CABOT: That is true, but it should not have prevented them from getting evidence of ascites. Of course a mesenteric thrombosis might possibly account for the board-like abdomen and hemorrhage, although I have never seen such a lesion cause hemorrhage. The advantage of that hypothesis is that we can make it a part of arteriosclerosis, which is one of the things I am seriously considering.

A STUDENT: In the presence of this thrill and of the large pulse pressure, in considering arteriosclerosis as the cause of his regurgitation is it not possible to assume that he had a moderate amount of stenosis?

DR. CABOT: The thrill is at the apex. He has no right to have it there from aortic stenosis. I do not know what to make of it. I have to throw it out.

The only thing that I feel clear about is the aortic regurgitation.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Luetic heart disease. Aortic regurgitation.
Luetic aortitis.
Congestive failure.

DR. RICHARD C. CABOT'S DIAGNOSIS

Aortic regurgitation.
Possible peptic ulcer.

ANATOMIC DIAGNOSES

1. *Primary or fatal lesion.*

Rheumatic heart disease. Aortic insufficiency.
Fibrous myocarditis.

2. *Secondary or terminal lesions.*

Bronchopneumonia.
Chronic passive congestion.
Ulcer of the stomach.

3. *Historical landmarks.*

Healed infarcts of the lung.
Angioma of the liver.
Hypertrophy of the prostate.

DR. TRACY B. MALLORY: The heart is most interesting here, so I shall start with that. It weighed 500 grams. The myocardium was greatly thickened and showed many small white scars. The aortic valve showed an increase in circumference to 9 centimeters—normally about 7 centimeters—and interadherence of the left and posterior cusps. The right and posterior cusps were slightly separated. This appearance left us in doubt as to the exact nature of the process. The remainder of the aorta showed a moderate degree of arteriosclerosis, nothing more. There was no definite evidence of syphilis anywhere in the aorta beyond the valve. Often on these aortic valves you can make a snap diagnosis in gross, as to whether they are syphilitic or rheumatic in origin, by noting interadherence or separation of the cusps. Normally the edges of the cusps come back to a single point-like contact at the connection with the aorta. The rheumatic type in general tends to draw the cusps together and make them adherent over a distance of one, two or three millimeters. Syphilis on the other hand generally tends to ulcerate away a little bit at the point where the cusps join the aorta, and consequently separate the margins. In this particular case one margin was drawn together and another was separated, so we are between two fires. We attempted to stain it for spirochetes, but it will be some time before we get a result on that. As for other evidence of syphilis in the body, the only suggestion we could find was a slight roughening and bowing of the tibia. I do not know whether that was noted in the clinical record or not. Elsewhere there was nothing to suggest lues.

The coronary artery showed a small number of raised atheromatous plaques which encroached slightly upon the lumen, but caused no marked degree of obstruction, and the orifices of the coronary arteries were not involved in the process that had affected the valves.

The lungs showed a terminal bronchopneumonia.

The liver was enlarged and showed an acute congestion on top of a slight degree of chronic passive congestion. It had not however the appearance of the liver of longstanding cardiac failure.

The kidneys were negative.

The remaining point of interest is the stomach. One centimeter from the pylorus was a large ulcer three and a half centimeters in diameter, indurated at its base and adherent posteriorly over the head of the pancreas in the immediate region of the gastroduodenal artery, which had been partially ulcerated through by the process. This undoubtedly was the source of his big hemorrhage. The degree of hemorrhage from a duodenal ulcer of course depends chiefly on whether a large vessel is involved in the process or not. The gastroduodenal is perhaps the commonest artery from which these ulcers bleed. Occasionally an ulcer on the greater curvature of the stomach will work down upon the celiac axis. I have seen fatal bleeding from this artery cause death in about an hour and a half.

A STUDENT: How do you account for the adenopathy?

DR. MALLORY: It certainly was not very marked post mortem. That is one sort of thing that every patient that you have in the hospital always shows, if you look for it. You always find enlarged inguinal glands and often axillary.

DR. CABOT: I would warn you again about how silent a peptic ulcer can be, even one that has ulcerated through.

A STUDENT: Do not a number of cases get a terminal ulcer?

DR. MALLORY: You do find shallow ulcers or rather erosions on the mucosa which are agonal, but not a large indurated, perforating type of ulcer like this. This was unquestionably an ulcer of weeks' to months' standing.

A STUDENT: Was there anything to account for that shadow that we see in the X-ray?

DR. MALLORY: We have no explanation for that. The heart was slightly dilated, but there was no aneurysm.

DR. CABOT: A world of mystery!

CASE 13292

A PROBLEM IN RENAL DIAGNOSIS

SURGICAL DEPARTMENT

A Canadian laborer forty-two years old entered the hospital for the first time March 19, two years before his final admission. The complaints were burning micturition, cloudy urine, bearing down pains and aching bladder and testicle.

Nine years before admission he noticed burning, high colored, foul smelling urine and some dribbling of thick material. The symptoms had persisted and during the past three years had been considerably worse. He urinated five to eight times a day and had inconstant nycturia two or three times a night with some urgency, occasional difficulty in starting the urinary stream and occasional shutting off of the stream due to burning in the bladder region. He sometimes had remissions of the symptoms and felt well for a week or two. For three years he had had right inguinal hernia.

Records of the Consultation Clinic show a visit November 16, four months before admission to the wards. On examination at that time the lower portion of the right kidney was felt with difficulty, not tender. Both epididymes were thickened and each showed a nodule measuring one-half to two centimeters on the lower pole. Vas normal on each side. Prostrate not enlarged but very tender. Both vesicles were felt, cord-like and tender. Secretion plentiful and showed 8 to 15 pus cells per oil field. Urine foul, cloudy and alkaline. Cystoscopy at this time showed edema and redness beginning in the middle of the trigone and extending laterally around the left ureteral orifice, where they were well marked. This area was very congested and bled easily. The right ureter was catheterized to the kidney without difficulty and drained clear urine which later became blood tinged from catheter trauma. The left orifice could not be found after prolonged search. No stones were felt with a stone searcher. The bladder urine was normal, cloudy, alkaline, no sugar, a very slight trace of albumin, sediment loaded with pus and bacteria, mostly cocci single and in chains, rare colon bacilli, occasional red cells. The right kidney urine showed a moderate number of red cells and bacteria, mostly single and in chains, a moderate number of epithelial cells, rare leucocytes. By X-ray the kidney outlines were obscured by many intestinal shadows. The opaque catheter did not pass above the upper portion of the sacrum. In the region of the prostate were a number of small shadows suggesting stones.

One sister of the patient died of tuberculosis. His wife had had one miscarriage.

As a child he had night sweats; of late years he had had only an occasional one. Twenty-four years before admission he had Neisser infection for nine months. Nine years before admission he had a swollen, painful left testicle for a month. For five years he had had hemorrhoids with occasional bleeding. Four years ago he had "bronchial asthma" for five months. For four years his feet had occasionally been swollen. He had occasional non-radiating precordial pain. For several years he had had cough with white frothy sputum. He caught cold easily. He used a pound of smoking and chewing tobacco and a pint of alcohol a week.

Clinical examination in the wards showed a well nourished, flushed, healthy looking man with a hacking cough, able to walk about. Musical and sonorous squeaks in the left upper lobe and apex on inspiration. Heart normal. Blood pressure 128/85. Right kidney palpable? Small right inguinal hernia. Slight tenderness over the bladder region on deep palpation. Tenderness in the midline just above the prostate at the base of the bladder. Prostate not enlarged. Both vesicles barely palpable. External hemorrhoids. Extremities, pupils and reflexes normal.

Urine 43 to 95 ounces, specific gravity 1.015 to 1.017, cloudy or muddy at all of three examinations, a very slight trace of albumin twice, sediment loaded with pus cells and showed 5 to 15 red cells at one examination, 50 to 100 leucocytes and rare red cells at another. Renal function 90 per cent. Blood not recorded. Wassermann negative. Non-protein nitrogen 36.9.

The chart was not remarkable except for a rise of temperature to 100.9° on March 24 and of pulse to 100 the following day.

March 22 cystoscopy was attempted under local anesthesia, which was unsatisfactory. X-ray examination two days later gave somewhat better plates than those taken for the Consultation Clinic, although there were still numerous intes-

tinal shadows present and neither kidney outline was made out. On the left, scattered throughout the region from the level of the tenth rib to the level of the third lumbar vertebra, were numerous small dense rounded shadows. On review of the previous plates similarly distributed shadows were seen, somewhat obscured by motion but apparently the same shadows. Two or three similar small shadows were seen on the right between the eleventh and twelfth ribs. These were unlike renal shadows both in character and distribution. Their appearance was very unusual and could not be positively identified. In the plate of the lower portion of the tract there was a group of small shot-like shadows low in the pelvis seen within an area of gas in the rectum. No characteristic shadows of stone were seen in the genito-urinary tract.

March 27 a second cystoscopy was attempted under spinal anesthesia. This also proved a failure, and was the most painful experience the patient had had. April 1 a third cystoscopy was done, and a right pyelogram. April 5 the patient was discharged.

History of interval. For the next nine months he felt better than he had for several years. He was able to work steadily, and gained some weight. In the middle of November, three weeks before his second admission, his testicles began to swell and he had pains and aches in the legs with muscular weakness, marked frequency and difficulty in starting the urinary stream. He felt cold all the time. He had some pain in the left side of the back and the flank. These symptoms lasted for several days, gradually subsiding. At the time of his second admission he felt well except that his testicles ached and he felt pressure on the bladder.

Second admission, December 8, eight months after his discharge.

Clinical examination as before except as noted. Inguinal glands shotty and discrete. Some tenderness in the left costovertebral angle and on deep pressure in the left flank. By rectal examination prostate tender. Both epididymes swollen. A nodule at the superior pole on the right.

Urine 65 to 150 ounces until December 21, then amount normal until December 27, then 68 to 143 ounces; specific gravity 1.010 at the only test, cloudy or hazy at 6 examinations, a very slight trace to a trace of albumin twice. Sediment from the right kidney showed rare leucocytes; 4 of 6 other specimens showed leucocytes, 2 loaded with leucocytes or showed clumps of pus, one after cystoscopy December 18 loaded with red cells, no leucocytes. Renal function December 8: appearance time 6 minutes, in two hours 25 per cent. Blood not recorded. Wassermann negative. Non-protein nitrogen 40 milligrams. Uric acid 4.6 milligrams.

X-ray December 10 showed the right kidney rather large and low, its outline not abnormal. The upper pole of the left kidney could be made out, although the lower portion was obscured. Numerous dense shadows on the left side seemed to overlie the kidney and extended well up toward the diaphragm outside the kidney region.

On admission temperature 100°, pulse 95, respiration 32. Afterwards chart normal until after operation.

December 12 cystoscopy was done, and another right pyelogram.

December 17 operation was done. In ten days there was practically no drainage from the wound and the patient was urinating normally. A number 28 sound was passed with much pain but without much difficulty. January 2 he was discharged.

History of interval. For three months after leaving the hospital he did not feel well and had a good deal of pain in the area of the incision. After that he improved and during the summer felt well. In the autumn he had three or four attacks of pain in the right side and passed urine with a heavy sediment. Early in March, three weeks before his third admission, he had an attack of severe pain in the right flank, with vomiting, and passed no urine for thirty-six hours. March 22 he was again seized with sharp pain in the right flank, vomited steadily, and passed no urine. He had headache, vertigo and blurred vision.

Third admission. March 25, fifteen months after his last discharge.

On clinical examination he was flushed and in great distress. Breath ammoniacal. Skin dry. In the right flank extending out from under the lower border of the ribs a large tender mass was palpable, moving on respiration, thought probably to be the kidney. No kidney could be felt on the left. Prostate firm but not hard or enlarged.

Temperature 101.4° to 99°. Pulse 92 to 150. Respiration 24 to 33.

No laboratory examinations were made.

X-ray showed the kidney shadows obscured by considerable gas, especially on the left. The upper pole of the right was faintly outlined. This kidney appeared larger than usual. Just lateral to the transverse process of the second lumbar vertebra was a rounded shadow nearly a centimeter in diameter. There was some gas in the surrounding regions and it was thought possible that it might represent a bowel shadow. There was also a small area of increased density about an inch above the iliac crest, possibly within the kidney, a similar shadow below the eleventh rib in the right kidney area, and a smaller shadow between the shadows of the eleventh and twelfth ribs. Medial to the right ischial spine was a small shadow rather suggesting a phlebolith. There were multiple small shadows in the upper left quadrant which suggested multiple calcified areas in the spleen.

March 26 cystoscopy was done, followed by

operation. The patient did not survive the operation.

Discussion

BY HORACE K. SOWLES, M.D.

This is a very long history of some urinary infection.

"The left ureteral orifice could not be found after prolonged search." That probably means that there was so much edema and inflammation around the orifice that it could not be located.

We have here a few perfectly definite things. He undoubtedly has an epididymitis. There are as a rule only two infections which are apt to cause a chronic epididymitis. One is tuberculosis and the other is gonococcus. He has a very definite cystitis, and he has, so far as our information goes, indications that that cystitis is more marked around the left ureteral orifice, which leads us to the deduction that possibly the cystitis is caused from above, and that he has pathology in the left kidney. The right kidney is practically normal.

One sister died of tuberculosis. That immediately recalls our attention to his genito-urinary findings. We begin to wonder whether they might be of tuberculous origin.

Night sweats are another point slightly suggestive, but probably meaning nothing in the normal person.

The Neisser infection is a possible explanation for his epididymitis, but that is a pretty long time ago, and I should say that probably his present epididymitis is not a survival of that infection.

His swollen feet, his "bronchial asthma," and his precordial pain do not stimulate us very much in regard to the rest of his story. His cough may be suggestive. "He caught cold easily." That may be suggestive.

The renal function is normal. In spite of a possible left kidney disease, one kidney or the other is holding up its function pretty well.

The non-protein nitrogen is within normal limits.

Cystoscopies are often unsatisfactory with an acute inflammatory process in the bladder. The patients are so sensitive that it is practically impossible to do the examination under local anesthesia. We have no definite proofs that those shadows are in the kidney, although they may possibly be. They are more likely to be, perhaps, retroperitoneal glands. The X-ray people evidently feel that there is nothing characteristic of stone along the genito-urinary tract.

The failure of the second cystoscopy may have been due to incomplete spinal anesthesia. Good spinal anesthesia ought to give good bladder anesthesia. It was the most painful experience the patient had.

We can say very definitely that he has a definite cystitis. It was suggested, at least, by the first cystoscopy, that he has a left kidney pathology. We know definitely that he has an epididymitis. It is definitely stated that he has an inflammation of the vesicles, and he had quite a painful prostate on one examination. That has subsided on another examination. I should say we could state that he has a definite inflammation of his whole genito-urinary tract, and I think we almost come down to tuberculosis from the present findings.

There is one condition which might give such a painful bladder, but there is no suggestion of its proof in the cystoscopy. It is suggested slightly by his alkaline urine and by his very sensitive bladder, and that is the condition known under different names, characterized by alkaline urine and phosphatic encrustations of the mucous membrane. But the first cystoscopy made no note of any such deposit as that on the bladder walls. It was more a cystitis and not an encrustation. It was too long a story for a tuberculosis, but I am beginning to think that that was the answer.

First cystoscopy, March 22, first admission

No preliminary diagnosis is recorded. Under local anesthesia the right ureter was seen, the left not seen. The anesthesia was unsatisfactory.

Second cystoscopy, March 27, first admission

No preliminary diagnosis is recorded. Sacral novocain. The right ureter was easily seen. Prolonged search for the left ureter was unsuccessful. There was one spot in the edema of the left side which looked like the ureter, but no catheter could be made to enter it.

Third cystoscopy, April 1, first admission

No preliminary diagnosis is recorded. Spinal anesthesia. The right ureter was easily seen and was catheterized. The left ureter was not found.

Right pyelogram

The outline of the upper two-thirds of the kidney was visible. The lower pole was obscured by intestinal shadows. The kidney appeared rather large. The outline of the left kidney was not made out. The injected pelvis of the right kidney was large and appeared irregular or distorted. The character of the irregularity suggested rotation of the kidney, and the shadow being foreshortened some of the calices were rather blunt. The cupping, however, appeared to be preserved. The course of the ureter diverged somewhat more than usual from the midline and passed the crest of the ilium. When the catheter was withdrawn the ureter appeared normal down to a point just below the lower border of the sacro-iliac junction, where there was a slight irregularity suggestive of spasm or possibly partial constriction. This however appeared to offer no obstruction. No shadows suggesting stones were seen along the course of

the ureter or in the kidney region. The many small shadows previously described in the left upper quadrant were still present. A few similar small shadows were visible in the right upper quadrant.

Further Discussion

The obscurity surrounding the left kidney is still present. The right kidney, except for possible torsion, I should say does not give us much information.

He ought not to get a remission in a tuberculous genito-urinary infection. It is usually progressively downhill, and this whole story is rather long for a tuberculous infection. People do not usually survive nine years with such severe urinary symptoms. Yet I can hardly believe that it is possible to have such an extensive pyogenic infection with destruction of one kidney possibly discharging pus down into the bladder, causing a cystitis there. I cannot quite believe that this is a pyogenic infection, because he would get much more general symptoms from an extensive infection of that sort, and as a rule less marked bladder symptoms. An extensive bladder inflammation and blood bring us back again to tuberculosis.

There appears to be a pretty definite remission of symptoms here for a period of nine months, which is rather unusual.

At the second admission the specific gravity of the urine is rather low, but he is passing a good deal of urine and probably taking a good deal of fluid. He has had a definite decline in kidney function from ninety per cent. down to twenty-five per cent. in two hours. The uric acid is high.

We get a suggestion here of involvement of the right kidney perhaps. The urine is not so normal as it was on previous examinations, and there is a definite decrease in the kidney function.

Again they could not see the left kidney by X-ray. I do not think we have any additional information except that we now have evidence that the right kidney is becoming involved as well as the left.

Preliminary Diagnosis December 12, Second Admission

Prostatic calculi.
Renal stone?

Fourth Cystoscopy

The right ureteral orifice was easily located and the ureter catheterized without difficulty. Prolonged search failed to reveal any ureteral orifice or even any parts of the ureteral ridge on the left side. The mucosa in this region was normal in appearance. There was no evidence that the ridge or ureter had been obscured by any pathology. Also after long search a second orifice could not be found on the right. The prostatic ring was somewhat congested and instrumentation caused slight oozing from the mucosa into the prostatic portion in the urethra. This was apparently a case of a solitary kidney.

Further Discussion

The description here suggests possibly an anatomical abnormality. The mucosa in this region is said to be normal in appearance. If it is normal in appearance they should be able to find the orifice. There is said to be no ureteral ridge. That suggests a possible anatomical deviation.

There is very definite information which discounts definitely the assumption which we have been going on, that in the presence of a severe cystitis they could not find the ureteral orifice. This seems to point out that there is no ureteral orifice, because the mucous membrane is normal, that it is an anatomical freak of some sort, and that calls us back to our previous pyelogram, which said that the pelvis of the kidney was distorted. That brings us to an anatomical distortion and the question whether it may be a polycystic right kidney. That gives a very different tack to argue on than the one we have been going on to this point.

During the interval between the second and third entries he had a definite suppression, perhaps some uremia.

At the third admission he was parched, acidotic. No kidney felt on the left is in line with our latest deductions.

To my mind there are two definite trains of thought on this problem. My original idea was a genito-urinary tuberculosis involving all the way through the epididymis, vesicles, bladder, kidney, the left particularly. Then we come to the suggestion of the third cystoscopy, which is very definitely suggestive of an abnormal solitary kidney, enlarged, distorted, perhaps polycystic. But I cannot quite make that anatomical deviation account for all his severe infection and his epididymitis and vesiculitis. I believe he has probably a solitary and possibly an infected kidney, probably partially destroyed. But I believe also that he has a genito-urinary tuberculosis, although his epididymitis and his vesiculitis may be a hangover from his twenty-four-year-old gonococcus infection,—a possibility I should not expect.

X-Ray Interpretation of Pyelogram Taken April 1, First Admission

The general appearance is that of some displacement of the right kidney and ureter by a possible mass outside of the kidney area rather than of pathology in the kidney itself.

Pre-Operative Diagnosis December 17, Second Admission

Prostatic calculi.

FIRST OPERATION

Spinal novol. Lithotomy position. Median perineal incision. The bulb was dissected up and the prostatic urethra opened. With a sound in the urethra and a finger in the rectum a thickened area was found just to the right of the median line but no definite crepitus obtained. On inserting the finger into the prostatic urethra there was found a cavity about the size of a peanut on its floor containing fifteen small dark stones, apparently hard, each with many facets, one stone about the size of a pea and the others the size of a kernel of wheat. The prostate was apparently not enlarged, but the bladder orifice seemed a little tight. A soft rubber catheter was passed into the bladder through the perineal wound.

FURTHER DISCUSSION

Evidently there was an infection in the prostate which was keeping up bladder irritation and cystitis. As the history goes the only clue we had to that was on two occasions a sensitive, tender, but never an enlarged prostate.

This time, I suspect, they attempted to do something to the right kidney, but I should feel that it was impossible to do anything except a pyelotomy, to allow a freer drainage to overcome his uremic condition.

PRELIMINARY DIAGNOSIS MARCH 26, THIRD ADMISSION

Anuria.

FIFTH CYSTOSCOPY

Right ureter catheterized. The catheter seemed to pass well up into the renal pelvis. No urine was obtained.

ADDITIONAL NOTE FROM HISTORY

After the attack three weeks before admission the patient passed several small stones.

SECOND OPERATION

Gas-oxygen. Oblique right lumbar incision. The subcutaneous tissues were edematous, the perirenal fat dense, adherent, edematous. A very large, fairly movable kidney was found which it was impossible to deliver owing to the abnormality of its pedicle and its large size. The kidney was decapsulated, showing innumerable yellow elevated abscesses about the size of a pin head dotted over its surface. The ureter was not identified and the pedicle was not investigated owing to the fact that the patient went into collapse. The operation had to be stopped for a time until his condition could be improved. A clamp was then placed through the cortex of the kidney in such a way as to enter the pelvis, but no retained urine could be demonstrated. The patient was then reported in extremely poor condition and the operation was stopped. The wound was hurriedly closed with three through-and-through silk-worm-gut sutures. Before this was completed the patient ceased to breathe and no pulse could be obtained. No response could be obtained after long artificial respiration.

FURTHER DISCUSSION

Evidently it was a chronic pyonephrotic kidney. I should feel that that operation was merely a desperate chance, and probably the operator did not entertain any hope of benefitting the patient,—merely took a desperate chance that perhaps decapsulation and pyelotomy, by allowing freer drainage, might have an influence on the evidently uremic state which the patient was in.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Pyelonephrosis.
Anuria.
Uremia.

DR. HORACE K. SOWLES' DIAGNOSIS

Chronic pyonephrosis of the right kidney.
Absence of the left kidney.
Prostatic calculi.

ANATOMIC DIAGNOSES

1. *Primary fatal lesions.*

Pyonephrosis and pyelonephritis, right.
Right ureteritis with obstructing stone.
Anomaly: one kidney, and that the right.

2. *Secondary or terminal lesions.*

Edema of the lungs.
Soft hyperplastic spleen.

3. *Historical landmarks.*

Operation wounds.

DR. RICHARDSON: He was a well nourished white man. There was the operation wound as mentioned and an old scar in the perineum. The organs above the diaphragm call for no note except that the heart was rather large, 380 grams, but the valves and cavities were negative. The vascular apparatus elsewhere was in good condition. The liver, spleen and adrenals were negative. There was a right adrenal and a left adrenal, but no left kidney and no vestiges of it, or of the ureter, and no opening into the bladder.

The right kidney was present and weighed 665 grams. Its shape was that of a thick crescent. The capsule had been stripped. The surface was fairly smooth but dotted over with small abscesses. The substance of the organ was sown with smaller and larger abscesses and yellowish streaks and areas. This kidney had but one pelvis, which was dilated and contained much semifluid purulent material and several small

fibrinopurulent felt-like masses. We see those once in a while in dilated pelves, where the material presents this peculiar shape and consitency, something like thick plaques and ovoid masses, at times so rough and granular that they look at first like stones, which they are not. We had one case where the pelvis was filled with them and they looked very much like stones.

The right ureter was present and slightly dilated. The mucosa was negative except in the region of its junction with the pelvic mucosa of the right kidney, where it showed some erosion and coating with fibrinopurulent material. At this point the ureter was obstructed by a small, roughly spherical, hard stone with a pale granular surface, and this was wadded around by felty purulent material. All together it completely obstructed the ureter.

The bladder was negative, with only one ureteral opening and that the right one. The prostate was negative except that the prostatic urethra was large, and in the region of its posterior wall, lower half, there was an oval depressed area 2½ centimeters by 1 centimeter the base of which was smooth and clean and the margins smooth, rounded and slightly overhung. It looked like a healed lesion.

The seminal vesicles and testes were negative.

NOTES ON THE WORK OF DR. ALICE HAMILTON TAKEN FROM ACCOUNTS IN THE DAILY PAPERS

Dr. Hamilton of Harvard Leads in War on Industrial Diseases

Workers in the metal trades, where extremely dangerous industrial poisons sap the vitality of the employee, have a notion in the back of their heads that a scientist, a test-tube in hand, is fighting their common enemy. They are partly right.

One of the internationally famous leaders in this warfare is a woman—Dr. Alice Hamilton, first woman to be a member of any Harvard faculty, and now assistant professor of industrial medicine at the Harvard Medical School.

EAGER TO PROTECT WORKERS

Dr. Hamilton strongly endorses the belief that American manufacturers are willing to incorporate every device possible for the protection of their workers and even seek scientific advice on the subject.

Dr. Hamilton has been actively engaged since 1910 in research and protective measures concerning industrial hygiene. Her interest in this all important subject came about in the casual picking up of a book, an English book, "Dangerous Trades," by Thomas Oliver.

An opportunity to study this question came in 1910, when she was appointed a member of a commission to study occupational diseases in Illinois. A survey of all the industries using lead and arsenic in that state was commenced.

That fall she attended a meeting of the international congress for industrial hygiene in Brussels, and there it was made clear that our country was far behind all the rest of the civilized world in our knowledge of the dangers of industry.

The United States government awoke to this fact and asked her to make a series of investigations of the poisonous trades, especially lead, for the Department of Labor. This was the beginning of a work for the government which lasted until 1921, and during which time she studied such industries as smelting lead, making white lead, glazing and decorating pottery, the painter's trade, the printer's trade, the making of storage batteries, the enamelling of bathtubs and sinks, and the compounding of rubber goods.

In the spring of 1916 it became evident that the manufacture of explosives for the allies was attended with much danger to the workmen. From that time until the signing of the armistice it was her duty to visit plants making explosives and loading shells, and also airplane plants which were using poisonous dope on the planes.

After the armistice the new and largely developed aniline dye-works remained to be studied.

Now investigation of carbon monoxide poisoning in steel mills, coal mines, and the making of illuminating gas, mercurial poisoning in the making of felt hats and in the mining and reduction of mercurial ore.

Dr. Hamilton was born and brought up in Fort Wayne, Ind.; educated at home until she was 17 and then went to Miss Porter's school in Farmington, Conn.

When she was 21 she went to the medical school of the University of Michigan, a co-educational institution, and two years later, when she graduated, she went abroad to the Universities of Leipsig and Munich. Her first position was as professor in the Woman's Medical College of Northwestern University in Chicago.

It was in 1902 that Dr. Hamilton attracted the attention of the medical world. She had been working with the Memorial Institute for the study of infectious diseases in Chicago when an epidemic of typhoid fever swept through the tenement districts and her report, entitled "Flies, Filth and Fever," startled the entire profession.

During this time Dr. Hamilton had been living at Hull House in Chicago, and through her entire career she has devoted a certain part of each year to that institution. Jane Addams is one of her most devoted friends and co-workers.

Aside from the remarkable strides in her profession, in spite of the fact that she is the first woman to sit in the council chamber of Harvard, Dr. Hamilton is a gracious person. In the lecture hall she is vivid, and she has the added advantage of a keen, intuitive tact and a sense of humor.

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THE MONEY VALUE OF HUMAN LIFE

IT is perhaps surprising that human life should have existed for so many thousands of years as the chief factor in our world, with so little attention paid to its value. The appreciation of the actual worth of a life from the economic viewpoint is doubtless a development of civilization. It is true that attempts have been made from time to time to estimate in terms of currency the value of a life; Hammurabi (2123-2081 B. C.) assigned a definite value to every man, woman and child in his kingdom, but this conception lost its significance until the early middle ages, when the ancient Germans, Franks and Anglo-Saxons revived it in the form of "wergeld," or compensation for loss of life through murder. Edward the Confessor (1041-1066) published a list of life values, but the question was not scientifically studied until Sir William Petty, in 1690, discussed it in his "Political Arithmetick." Since that time numerous economists have written upon this subject. The most recent book dealing with the money value of human life is "America's Human Wealth," by Edward A. Woods and Clarence B. Metzger.*

These authors attempt to estimate the money value of the population of the United States, basing their conclusions on the earning power of the nation. They approach the problem in several ways, checking one result against the other, and by averaging the estimates so reached, they conclude that the value of the population in 1927 exceeds sixteen hundred and fifty billion dollars ($1,650,000,000,000). They point out that the economic value of man increases with his longevity; in 1900 the average length of life was between 45 and 48 years, whereas the expectation of life for a child born in 1926 is estimated at 58 years. The value of the increased earning power of the population for 1924 as compared with that of 1901 has been estimated at three and a half billion dollars per annum. The moral of this is evident: to increase our potential wealth (at least that portion due to human effort as opposed to material wealth) we must prolong life.

Woods and Metzger, whose book, by the way, is "approved by the National Association of Life Underwriters and the Life Underwriters Association of Canada," point out that this vast human treasure is protected only to a very limited degree by life insurance. In this country the life insurance carried by the population amounts to only 4% of the estimated value of that population. The amount of insurance carried has increased tremendously since 1912; at the end of 1926, the legal reserve companies alone had over $82,000,000,000 of insurance in force, covering at least 60 million lives. From the point of view of the policy holders in the life insurance companies, an increase in longevity is again greatly to be desired. For each year that the annual death claims can be deferred, over $40,000,000 will be saved to policy holders.

The medical profession, as one of the most important agencies in promoting longevity, is thus seen to be a factor of weight in upbuilding the wealth and prosperity of the nation. Perhaps some day there will appear a supereconomist who will deduce from all these figures the value to the community in dollars and cents of the medical profession. Given the actual worth of our services in terms of years, population and dollars, we can perhaps work out a fee table based upon something more tangible than is the present schedule of charges. Another possibility, equally improbable, is that a monetary value may be put upon the time doctors spend in free clinics, thereby enabling the medical men to deduct this amount from their income tax returns under the heading "gifts to charity."

*F. S. Crofts & Co., Publishers. 193 pages.

THE PASTEURIZATION OF MILK IN MASSACHUSETTS

THE care which is exercised by the Massachusetts Department of Public Health in preparing its regulations is in all probability not appreciated by the layman and probably but partially

so by the professional. A running sketch, therefore, of the procedure of the department in the forthcoming regulations with reference to the pasteurization of milk may be useful in an informatory way. It will be seen that much more is involved than the mere throwing together of a set of rules, but that the utmost care has been exercised to make these sensible, workable and effective.

An Act, approved April 15, authorized, in fact made it necessary for the Department to issue regulations relative to the licensing of establishments for the Pasteurizing of milk. Work was begun at once on the framing of the regulations, and at the meeting of the Massachusetts Tuberculosis League on April 26, and the meeting of the Massachusetts Associations of Boards of Health immediately thereafter, Dr. Bigelow, State Health Commissioner, invited all who were interested to hearings at the State House in the matter.

The first of these hearings, on May 20, brought together more than fifty members of boards of health, to whom a tentative set of regulations was presented by Dr. Bigelow, acting as chairman, assisted by Mr. Lythgoe, head of the division of food and drugs. The chairman read the items of the proposed regulations, each being fully and freely discussed from the health administration point of view. Informal votes were frequently taken to show the sense of the meeting. Nearly three hours were consumed in considering the more important phases of the regulations. A large committee was appointed for further consideration of the matter.

On Thursday, May 26, there was a hearing before the milk contractors, some 75 being present. The order of procedure was the same as before, there was free discussion, informal votes were taken, and a committee of this group was named. This committee by itself and also with the committee of the health officers reviewed the whole matter, and the result was made up in manifolded form for presentation before the third hearing, that of June 7, at which both health officers and milk producers were present to the number of more than one hundred altogether. Again there was full discussion of the paragraphs in detail, and following it a meeting of the two committees was held to make use of any suggestion coming from the third hearing.

The matter thus refined by much discussion was on June 14 taken up by the Public Health Council, which after consideration referred it to the Food and Drug Committee of the Department, which early in July held conferences with interested groups in Boston and in Springfield. The final draft as approved by the Public Health Council, is that which has become the set of regulations under the Act, the time when they have become effective being by law, July 19.

It is not necessary here to print the regulations, since they have been supplied from the State House to all parties directly interested, but the evidences of most careful consideration on the part of the authorities, are of educational value, and should be known. At the hearings the State Commissioner of Public Health, leading the meeting skilfully through possibly controversial situations, and was ably seconded by Mr. Herman C. Lythgoe, head of the Division of Foods and Drugs.

Following closely upon the adoption of the regulations will come the meeting in Worcester of the Massachusetts Association of Boards of Health, at which "Positive Pasteurization" will be the principal subject for discussion.

THIS WEEK'S ISSUE

CONTAINS articles by the following authors:

BALDWIN, EDWARD R., M.A.; M.D. Yale Medical School 1890; Director Trudeau Foundation, Saranac Lake, New York. His subject is: "Specific Medication in Tuberculosis with Special Reference to Chemotherapy." Page 79. Address: Saranac Lake, New York.

HEAD, JEROME R., A.B.; A.M.; M.D. Harvard Medical School 1922; Instructor in Surgery University of Illinois. His subject is: "The Effect of Operation Upon the Vital Capacity." Page 83. Address: 122 S. Michigan Avenue, Chicago, Illinois.

WHEATLEY, FRANK E., A.B.; M.D. Tufts College Medical School 1914; Assistant Professor Roentgenology Tufts Medical School; Roentgenologist to Milton and Goddard Hospitals. His subject is: "Report of a Case of Eventration of the Left Diaphragm." Page 87. Address: 520 Beacon Street, Boston.

HUTCHINS, CHARLES P., M.D. Long Island College Hospital 1897; Former Professor of Physical Education, Dickinson College, University of Wisconsin and Indiana University; Director of Rehabilitation Clinic, Aetna Life Insurance Company. His subject is: "Treatment of Ankle Sprains." Page 91. Address: P. O. Drawer 38, Syracuse, New York.

HILL, LEWIS W., A.B.; M.D. Harvard Medical School 1913; Instructor in Pediatrics, Harvard Medical School; Assistant Visiting Physician, Children's Hospital; Visiting Physician, Boston Nursery for Blind Babies. His subject is: "Relationship of the Weather to the Incidence of Pneumonia in the Children's Hospital, Boston." Page 93. Address: 483 Beacon Street, Boston.

MYERSON, ABRAHAM, M.D. Tufts College Medical School 1908; Professor Neurology Tufts College Medical School; Visiting Neurologist, Boston City Hospital and Beth Israel Hospital; Psychiatrist Massachusetts Commission of Mental Diseases in Research Concerning Mental State of Prisoners. His subject is: "Progress in Neurology." Page 95. Address: 483 Beacon Street, Boston, Mass.

MISCELLANY

ELECTION OF DR. CHARLES P. SYLVESTER AS CHAIRMAN OF THE BOARD OF REGISTRATION IN MEDICINE

At the annual meeting of the Board of Registration in Medicine held July 14, 1927, Dr. Sylvester was elected Chairman; Dr. Birnie, former Chairman, having tendered his resignation from the Board to take effect July 15, 1927

Dr. Vaughan was re-elected as Secretary.

Dr. Sylvester has been a member of the Board since July, 1925, and has shown great interest in the problems of medical registration.

THE FORTY-SECOND ANNUAL MEDICAL REPORT OF THE TRUDEAU SANATORIUM

The annual report of the Medical Board, written by Lawrence Brown, in addition to discussing many questions of interest to those who are familiar with the work of the Sanatorium, includes many observations of a more general application. In speaking of the work in general, Dr. Brown says that "on the whole the type of cases admitted has been more advanced but the number discharged as apparently arrested has increased. The reasons for this are larger treatment and, thanks to the coöperation of the clinical and X-ray laboratories, complications and slight relapses formerly overlooked for some time have now been discovered at their onset.

The following paragraph dealing with the value of ultraviolet light and heliotherapy will interest and perhaps disappoint some of the enthusiasts.

"We have convinced ourselves that certain complications of pulmonary tuberculosis are definitely cured by the use of the 'ultraviolet ray' lamps but again we are not certain that we have not been overdosing some patients in our efforts to make sure of recovery. This problem has been and will continue to be followed. It is of interest to note that a number of cases of intestinal tuberculosis have been diagnosed before any symptoms have appeared and have ultimately recovered. The condition is found in about six per cent of all cases admitted. Investigation with other rays are under way in the research laboratory. Up to this time no one as far as we are aware has presented convincing evidence that heliotherapy has any beneficial influence upon uncomplicated pulmonary tuberculosis, but the day may come when we may have to use the roof of the Ludington Memorial or of the Gillender Cottage for this purpose."

In regard to the artificial production of immunity against tuberculosis Dr. Brown says:

"The main work of the research department during the year has been the further study of immunity in tuberculosis. Many attempts have been made to produce immunity in normal guinea-pigs by immunizing them with dead tubercle bacilli, and the conclusion was reached that a considerable amount of resistance could be established. However, before a definite conclusion can be drawn we must revise some of our methods in the study of such problems.

Dr. Petroff thinks that in practically all experiments in immunity up to this time too large doses of tubercle bacilli for testing the immunity have been injected, so large in fact that in many instances the immunity must be very pronounced in order to be detected. He has repeatedly shown that even five tubercle bacilli can produce tuberculosis in a normal guinea-pig. It has been usual in all experimental work in immunity in tuberculosis to weigh the dose of tubercle bacilli and as many as 80,000 to 100,000 bacilli have been injected to test the immunity. Dr. F. B. Jennings, together with Dr. Petroff, has devised an ingenious method of counting the tubercle bacilli and so accurately determining the number of bacilli in any given dose. This method should prove of much value in immunological work."

A summary of the present condition of all discharged patients states that of 6275 cases, of which 1775 (28.29%) were incipient, 3961 (63.12%) moderately advanced, and 539 (8.59%) far advanced, 2889 are well, 2393 are dead, 587 are living (but not well) and 406 are untraced.

The report also contains the tenth collection of the studies of the Edward L. Trudeau Foundation. These consist of *Immunological Studies in Tuberculosis,* by S. A. Petroff and F. W. Stewart; *A Modification of the Ziehl-Meelsen Staining Method for Tubercle Bacilli,* by F. B. Cooper; and *A New Method for Suspending and Counting Living Tubercle Bacilli,* by F. B. Jennings.

Two other short papers are published, one on the diagnostic and prognostic significance of pleurisy in pulmonary tuberculosis, by F. H. Heise and Laurason Brown, which closes with the statement that "Altogether it would seem that the occurrence of pleurisy with effusion exerts but little influence on the prognosis of any individual case of pulmonary tuberculosis for the first five years after discharge"; and one by F. H. Heise on the progression of lesions.

RECENT DEATHS

ALLEN—Dr. Maurice Joseph Allen of West Newton, a graduate of Harvard Medical School in the class of 1927, died at the Boston City Hospital, July 12, 1927, aged 32, from injuries received June 18 when a motor car in which he was riding hit an iron trolley pole at Pleasant and Stoughton Streets, Dorchester. Two other deaths have resulted from the crash. Only the driver of the car, Henry W. Boswell, restau-

rant manager at 240 Washington Street, escaped unhurt. Dr. Allen's home was Brooklyn, N. Y.

PITTINGER—Dr. Lee Ross Pittinger, a graduate of the College of Physicians and Surgeons, Boston, in 1907, died suddenly at his home in Allston, July 10, 1927, at the age of 52.

CORRESPONDENCE

A QUESTION OF RECOGNITION OF REPRESENTATIVES OF THE JEWISH RACE

July 8, 1927.

Editor, Boston Medical and Surgical Journal:

On page 1109 of the June 30th edition of the Journal I read with interest your editorial comment entitled "A Disgraceful Race Controversy." Your attitude is praiseworthy. But while we are on this subject, on page 1079 of the same edition is a list of sixty-one names of physicians appointed to serve as committeemen or delegates. Not one name was Jewish.

Very truly yours,
Bernard J. Klein, M.D.

Editorial Note:—The comment of our correspondent touches upon an important subject and it would be well if nominations could be made to include all who are interested in the affairs of the Society. If such claims have been made and ignored our Jewish members have ground for dissatisfaction. There are certainly many who could take on Society work creditably.

RECOMMENDATIONS RELATING TO THE CARE OF INFANTILE PARALYSIS, AND A LETTER TO BOARDS OF HEALTH

The Commonwealth of Massachusetts
Department of Public Health
State House, Boston

July 8, 1927.

Editor, Boston Medical and Surgical Journal:

I would be glad if you could find space to publish in an early number of your Journal this letter and the enclosure. The enclosure will be mailed to boards of health.

After consultation with members of the Harvard Infantile Paralysis Commission as well as several orthopedic specialists, it seems to me desirable to call the attention of the profession to the after care of cases of infantile paralysis.

The two principal considerations in the treatment of infantile paralysis are:

(1) To prevent deformities, and

(2) To secure as much recovery of muscle strength as possible.

I. PREVENTION OF DEFORMITIES

The main causes of deformity are:

Strong muscles outpulling weak ones,

Bearing weight on weakened muscles,

Contractures resulting from keeping the limbs or body in abnormal positions.

These deformities can be prevented by:

1. Maintaining the normal position of the limbs, which can be done best by the use of light splints, even in the early stage of the disease.

2. By complete rest in bed. Sitting up or walking should not be attempted, in many cases for as long as two or three months at least.

II. RECOVERY OF MUSCLE STRENGTH

Many muscles tend to recover strength under the regime of rest, while others require graduated exercises for the weakened muscles. However, no exercises or massage should be begun while there is tenderness or soreness. Tenderness or soreness may last for two or three months and can best be treated by complete rest, and warm baths or hot packs (temperature 105° F.).

Walking should not be allowed until the limbs and trunk can be maintained in their normal positions by their own muscle power or with the aid of braces.

Very truly yours,
George H. Bigelow, M.D.,
Commissioner of Public Health.

Commonwealth of Massachusetts
Department of Public Health

Infantile Paralysis (Anterior Poliomyelitis)

Although in the past severe epidemics of infantile paralysis have occurred in Massachusetts, the number of cases reported during the last five years has remained practically stationary. The average number of cases (1922-1926) has been 216. Past experience indicates that epidemics tend to follow several consecutive quiet years.

Since there is still uncertainty about the mode of spread of the disease, the exact measures for its control remain in doubt. For this reason the appearance of the disease causes unusual anxiety in a community. From what is known about the disease the following preventive measures can be sanely employed:

Patients with infantile paralysis should be isolated in the acute stage in much the same way as typhoid fever or diphtheria patients. It seems wise to treat all body discharges as infectious.

It is well to remember that healthy carriers or missed cases may play a large part in the dissemination of the disease. For this reason, contacts with cases might well be isolated for a time after exposure.

Since three authentic outbreaks spread by infected raw milk have been reported, it seems wise to protect against milk as well as other foods eaten raw. It would be absurd from this to look on milk as the only, or even the most common, method of spread of this disease, but it constitutes another argument for adequate pasteurization. This emphasizes the need of scrupulous cleanliness in handling food to be eaten raw.

The After Care of the Disease. After the acute symptoms of infantile paralysis have subsided, after the fever and pain and tenderness have gone, there usually remains some paralysis of the muscles. Sometimes this is so great that the child is helpless. Sometimes it is very slight, only weakness of a few muscles. Treatment should not be instituted while there is tenderness or soreness, neither should there be delay after these symptoms have disappeared.

Nearly every one of these children can be improved. In some the cure is almost perfect. It is not easy, however, to bring about improvement. It cannot be done by patent medicines, or street "fakers." It cannot be done by a careless physician who does not study the case thoroughly. It nearly always takes a long time. In most cases it is necessary to keep up the treatment for two years. Many times it should be kept up longer.

The parents of a child who has had this disease should keep it constantly under the care of an experienced physician. They should see that the physician's directions are carried out exactly. If unable to employ such a physician the child should be taken regularly to a clinic or dispensary.

Improvement is usually slow, but with proper treatment it is usually sure. Do not get discouraged. Keep up the fight. It would be sad indeed in future years for parents to feel that the infirmity of their child was in any degree due to their own carelessness or neglect.

DISEASE INCIDENCE IN CONNECTICUT
WEEK ENDING JUNE 25

DISEASE	1927					1926			
	Week ending June 4	Week ending June 11	Week ending June 18	Week ending June 25	Average cases reported for week corresponding to June 25 for past 7 years	Week ending June 5	Week ending June 11	Week ending June 18	Week ending June 26
Actinomycosis	-	1	-	-	-	-	-	-	-
Anthrax	-	-	-	-	-	-	-	-	-
Botulism	-	-	-	-	-	-	-	-	-
Cerebrospinal Men.	-	-	2	-	-	2	1	2	1
Chickenpox	114	123	123	90	29	54	58	92	39
Conjunctivitis, inf.	-	-	-	-	-	-	-	-	-
Diphtheria	26	31	28	34	30	18	9	15	13
Dysentery, Amob.	-	-	-	-	-	-	-	-	-
Dysentery, Bac.	-	-	-	-	-	-	-	-	-
Encephalitis, Epid.	1	1	-	-	1	-	1	-	1
Favus	-	-	-	-	-	-	-	1	-
German Measles	8	5	6	3	7	25	45	19	24
Hookworm Infection	-	-	-	-	-	-	-	-	-
Influenza	4	2	-	-	3	6	5	2	-
Leprosy	1	-	-	-	-	-	-	-	-
Malaria	-	1	1	1	1	-	-	-	-
Measles	44	67	55	68	155	421	444	349	244
Mumps	57	37	47	30	18	5	22	8	5
Paratyphoid Fever	-	-	-	-	-	-	-	-	-
Pneumonia, Broncho*	16	22	19	24	18*	24	30	24	24
Pneumonia, Lobar	20	25	33	23	15	25	39	38	26
Poliomyelitis	-	-	1	-	1	-	-	-	-
Scarlet Fever	68	89	56	48	46	80	79	78	49
Septic Sore Throat	1	5	2	6	-	-	-	1	-
Smallpox	-	-	-	-	-	-	1	-	1
Tetanus	-	-	-	-	-	-	-	-	1
Trachoma	1	-	-	-	-	-	-	-	-
Trichinosis	-	-	-	-	-	-	-	-	-
Tuberculosis	17	28	33	50	36	32	28	42	40
Tuberculosis (o.f.)	3	3	4	2	4	4	-	2	4
Typhoid Fever	-	2	1	-	6	1	3	3	1
Typhus	-	-	1	-	-	-	-	-	-
Whooping Cough	39	13	21	16	46	29	44	53	33
Gonorrhoea	67	24	28	55	14	5	45	9	11
Syphilis	29	29	21	24	23	13	58	17	12

*Average for two years. Made reportable January 1, 1925.

Remarks: No cases of cholera, Asiatic, glanders, plague, rabies in humans and yellow fever during the past seven years.

MASSACHUSETTS STATE DEPARTMENT OF HEALTH

Resume of Communicable Diseases June, 1927

GENERAL PREVALENCE

Disease prevalence for June was about normal.

More cases of poliomyelitis were reported for June than in any previous June since 1917. Mumps, chickenpox and lobar pneumonia were also reported in larger numbers than in any previous June on record. While scarlet fever is declining rapidly from the peak, it was still more prevalent this June than in any other June on record. The incidence of diphtheria, tuberculosis and influenza was about normal.

For the second month, typhoid fever reached a new low level.

No case of smallpox has been reported for fourteen months, the longest period without a case on record. The incidence of whooping cough and German measles was moderately low.

RARE DISEASES

Anterior poliomyelitis was reported from Boston, 3; Fall River, 1; Lee, 1; Malden, 1; North Attleboro, 1; Peabody, 1; Watertown, 1; Worcester, 2; total, 11.

Dog-bite requiring anti-rabic treatment was reported from Arlington, 4; Attleboro, 1; Billerica, 2; Boston, 6; Chelmsford, 1; Everett, 2; Holyoke, 1; Hudson, 1; Lexington, 1; Lowell, 8; Lynn, 3; Melrose, 1; Newton, 1; Norwell, 2; Quincy, 2; Revere, 4; Salem, 2; Somerville, 1; Springfield, 1; Taunton, 1; Weymouth, 2; total, 47.

Encephalitis lethargica was reported from Northampton, 1; Quincy, 1; Somerville, 1; total, 3.

Epidemic cerebrospinal meningitis was reported from Arlington, 1; Boston, 1; Cambridge, 1; Greenfield, 1; Holyoke, 1; Lynn, 1; Pittsfield, 1; Winthrop, 1; total, 8.

Malaria was reported from Lowell, 1.

Pellagra was reported from Holyoke, 1.

Septic sore throat was reported from Beverly, 1; Boston, 4; Brockton, 1; Cambridge, 1; total, 7.

Tetanus was reported from Amherst, 1; Fall River, 1; Northampton, 2; total, 4.

Trachoma was reported from Boston, 4; Fall River, 1; New Bedford, 1; total, 6.

MONTHLY REPORT OF CERTAIN COMMUNICABLE DISEASES

Diseases	Cases in entire population				Case rates per 100,000 population		
	June, 1927	June, 1926	Prosodemic index	Epidemic index	June, 1927	June, 1926	Expected rate‡
All causes	7,768	8,795	—	—	181.9	208.7	—
Anterior poliomyelitis	11	5	7*	1.6†	.2	.1	.1
Diphtheria	388	276	341*	1.1†	9.1	6.6	7.9
Measles	1,734	2,724	3,824*	.5†	40.6	64.6	89.6
Pneumonia, lobar	317	302	219*	1.4†	7.4	7.2	5.1
Scarlet fever	1,587	927	667*	2.5†	37.2	21.9	15.6
Tuberculosis, pulmonary	457	522	393*	1.1†	10.7	12.4	9.2
Typhoid fever	18	34	29*	.6†	.4	.8	.7
Whooping cough	406	776	733*	.6†	9.5	18.4	13.2
Chickenpox	874	708	—	—	20.5	16.8	—
German measles	79	1,023	—	—	1.9	24.3	—
Influenza	16	25	—	—	.4	.6	—
Mumps	1,044	600	—	—	24.5	14.2	—
Tuberculosis, other forms	94	89	—	—	2.2	2.1	—

*This index is an attempt to estimate the number of cases based on the trend during the past years which can be expected to occur, and is for the purpose of comparison with the number of cases which actually did occur.

†This ratio expresses how prevalent the disease is compared with the index mentioned above; 1.0 indicates that the actual number of cases equals the expected number. A larger number means a greater prevalence, and a smaller number a lesser prevalence than expected. Thus, 2.0 would indicate twice the expected number of cases, and .5 half the expected number of cases. The methods used to determine the index will be described in detail in an early issue of the JOURNAL.

‡Calculated from the Prosodemic Index.

NOTICES

REMOVAL

DR. JAMES B. THORNTON will be at 366 Commonwealth avenue, Boston, after the first of August.

UNION HOSPITAL IN FALL RIVER

THE Regular Monthly Clinical Staff Meeting will be held at the Country Club on Thursday, July 21, 1927, at 5:30 P. M.

M. N. TENNIS, M.D., *Secretary.*

A RULING OF THE INCOME TAX DEPARTMENT

THE compensation received by a medical director of a State or county hospital is no longer exempt from income taxes, under a ruling just announced by the Income Tax Department, according to M. L. Seidman, tax expert of Seidman & Seidman, Certified Public Accountants.

"Heretofore," Mr. Seidman explained, "the law used to be that a medical director in the position mentioned need not pay any income taxes on his compensation since he was a State employee or the employee of a subdivision of a State, and the salaries of such employees were exempt from tax. Under the 1926 law, the further requirement was added that the employment be in connection with an essential governmental function. It is now held by the Income Tax Department that operating a hospital is a proprietary rather than a governmental function, and hence the Department concludes that the compensation of a medical director is subject to tax under the present law.

"The probabilities are," Mr. Seidman added, "that this ruling will be contested, for there is a serious doubt whether conducting a hospital is purely proprietary. The contention may be made that the State wishes to safeguard life as well as property, and maintaining a public hospital could therefore be regarded as an essential governmental function."

BOOK REVIEWS

Principles and Practice of Oral Surgery. By S. L. SILVERMAN, D.D.S., F.A.C.D., Clinical Professor of Oral Surgery Atlanta-Southern Dental College; Associate Professor of Surgery (Oral) Emory University, School of Medicine; Formerly Special Lecturer on Oral Surgery Columbia University, New York; Oral Surgeon to the City (Grady) Hospital, Scottish Rite, Piedmont and St. Joseph Hospitals; Consulting Oral Surgeon to U. S. Veterans Hospital, Atlanta, Georgia.

This volume was written for beginners and the general practitioner. The author has attempted to cover a very extensive field in a small space of three hundred and twenty-two pages, and as a result, many of the subjects have received a bare mention. From our point of view it might be just as well to omit some of the chapters in a book of this kind, as they are not, strictly speaking, oral surgery, and allow a little more space for some of the vital subjects. The book contains very little original matter. The illustrations are good.